Vol. 67, No. 5 Established 1884 May 1950

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

E. B. TREAT & CO., Inc., *Publishers*, 45 East 17th Street, NEW YORK, 3
Yearly Subscription $6.00 (Foreign $6.75). Single Copy, $1.00

Entered as second-class matter Feb. 3, 1892, at New York, N. Y., P. O., under the Act of March 3, 1879

1. HIGH IN PROTEIN–13%–as a result, a single ounce of Cerevim provides 5½ grams of protein–plus:

2. THIAMINE–0.6 mg. per ounce of Cerevim "The cumulative effects throughout a lifetime...(of thiamine)...may spell the difference between alert, successful living and a marginal effectiveness."[1]–plus:

3. NIACINAMIDE–6.0 mg. per ounce of Cerevim in accord with The National Research Council's recommended allowance[2]–since "Nicotinic acid is found in natural foods only in limited amounts."[3]–plus:

4. RIBOFLAVIN–0.9 mg. per ounce of Cerevim for this factor is directly related to growth[4] and is "essential to the defense powers of the organism"[5]–plus:

5. CALCIUM–300 mg. per ounce of Cerevim thus supplying 3 times the calcium in a fluid ounce of milk–plus:

6. IRON–7.5 mg. per ounce of Cerevim since "a child's increasing need for iron cannot safely be left to chance."[6]–plus

7. COPPER–0.3 mg. per ounce of Cerevim in the 1:25 ratio which Elvehjem, et al.[7] and Cason[8] found particularly effective in raising hemoglobin levels in infancy.

With such natural foods of high biologic value as:

8. WHOLE WHEAT MEAL 9. OATMEAL 10. CORN MEAL 11. NON-FAT MILK SOLIDS 12. BARLEY 13. WHEAT GERM 14. BREWERS' DRIED YEAST 15. MALT

Leading to such benefits as the literature[9] reports:

16. "increase in urinary output of riboflavin" 17. "improvement in pediatricians' scores" 18. "improvement in skeletal maturity" 19. "improvement in skeletal mineralization" 20. "retardation of increase in dental caries" 21. "recession of corneal vascularization" 22. "improvement in the condition of the gums"

23. Better Bowel Function[10]

24. PALATABILITY–Cerevim makes all the above acceptable as well as available to infants and children.

in all–24 good reasons why CEREVIM is a first among first foods

a pre-cooked cereal for professional specification now produced exclusively at the M & R Dietetic Laboratories

BIBLIOGRAPHY:

(1) Harrell, R.F.: J. Nutrition 31:283 (Mar.) 1946.
(2) National Research Council, Recommended Dietary Allowances, 1945.
(3) McLester, J.S.: Nutrition and Diet in Health and Disease, ed. 4, Phila., W.B. Saunders Co., 1943, p. 78.
(4) Sherman, H.C.: Chemistry of Food and Nutrition, ed. 7, N.Y., Macmillan Co., 1946.
(5) Council on Pharmacy and Chemistry and Council on Foods of the A.M.A.: The Vitamins, Chicago, American Medical Association, 1919.
(6) Dickson, W.A.: Yearbook of Agriculture, U.S. Gov't Printing Office, Supt. of Documents, Washington, D.C., 1939, p. 203.
(7) Elvehjem, C.A., Siemers, A., and Hendemuth, D.R.: Am. J. Dis. Child. 50:28 (July) 1935.
(8) Cason, J.F.: J. Pediat. 4:614 (May) 1934.
(9) Urbach, C., Mack, P.B., and Stokes, J., Jr.: Pediatrics 1:70 (Jan.) 1948.
(10) Joslin, C.L., and Helms, S.T.: Arch. Pediat. 54:547 (Sept.) 1937.

reliable, convenient, versatile, palatable

3 new

water-soluble liquid vitamin preparations

Potent, economical and pleasant tasting, these three new vitamin preparations are ideally suited for routine supplementation of diets of infants, children and adults. ℂ They may be dropped directly into the mouth, stirred into the formula, or mixed into cereals or other solid foods. ℂ Each is supplied in 15 and 50 cc. bottles, with an appropriately calibrated dropper to assure accurate dosage and facilitate administration.

POLY-VI-SOL

Each 0.6 cc. supplies:

Vitamin A	5000 USP units
Vitamin D	1000 USP units
Ascorbic Acid	50.0 mg.
Thiamine	1.0 mg.
Riboflavin	0.8 mg.
Niacinamide	5.0 mg.

TRI-VI-SOL

Each 0.6 cc. supplies:

Vitamin A	5000 USP units
Vitamin D	1000 USP units
Ascorbic Acid	50 mg.

CE-VI-SOL

Each 0.5 cc. supplies:

Ascorbic Acid	50 mg.

MEAD'S

MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U. S. A.

TO CONTRIBUTORS AND CORRESPONDENTS OF

ARCHIVES OF PEDIATRICS

Subscription $6.00 a Year, in Advance **Foreign $6.75** **Single Copy, $1.00**

Editorial Communications address to JOHN F. LANDON, M.D., 120 East 75th Street, New York 21
Business Communications address to E. B. TREAT & Co., INC., 45 East 17th Street, New York 3

ORIGINAL ARTICLES, brief reports of rare and interesting cases, or new modes of treatment are solicited, but none will be considered for publication except with the distinct understanding that it is contributed exclusively to this journal. Manuscripts must be typewritten, double-spaced, and the original, not the carbon, copy submitted. The editor and publishers will not be responsible for views expressed.

ILLUSTRATIONS, as in the judgment of the editor are necessary, will be furnished free when satisfactory photographs or drawings are supplied. Photographs must be clear and distinct; drawings must be in India ink on white paper.

COPYRIGHT. — Original communications appearing in this journal are protected by copyright and can not be reproduced, either wholly or in part, without permission of the publishers.

REPRINTS of articles appearing among "Original Communications" may be ordered immediately upon receipt of galley proof by communicating direct with the publishers, E. B. Treat & Co., Inc., 45 East 17th Street, New York 3, who will supply their schedule of prices.

DISCONTINUANCES. — The publishers must be notified when a subscriber wishes his journal stopped and all arrearages must be paid. Without such notification it is assumed that a continuance is desired. Journals returned are not notice of discontinuance.

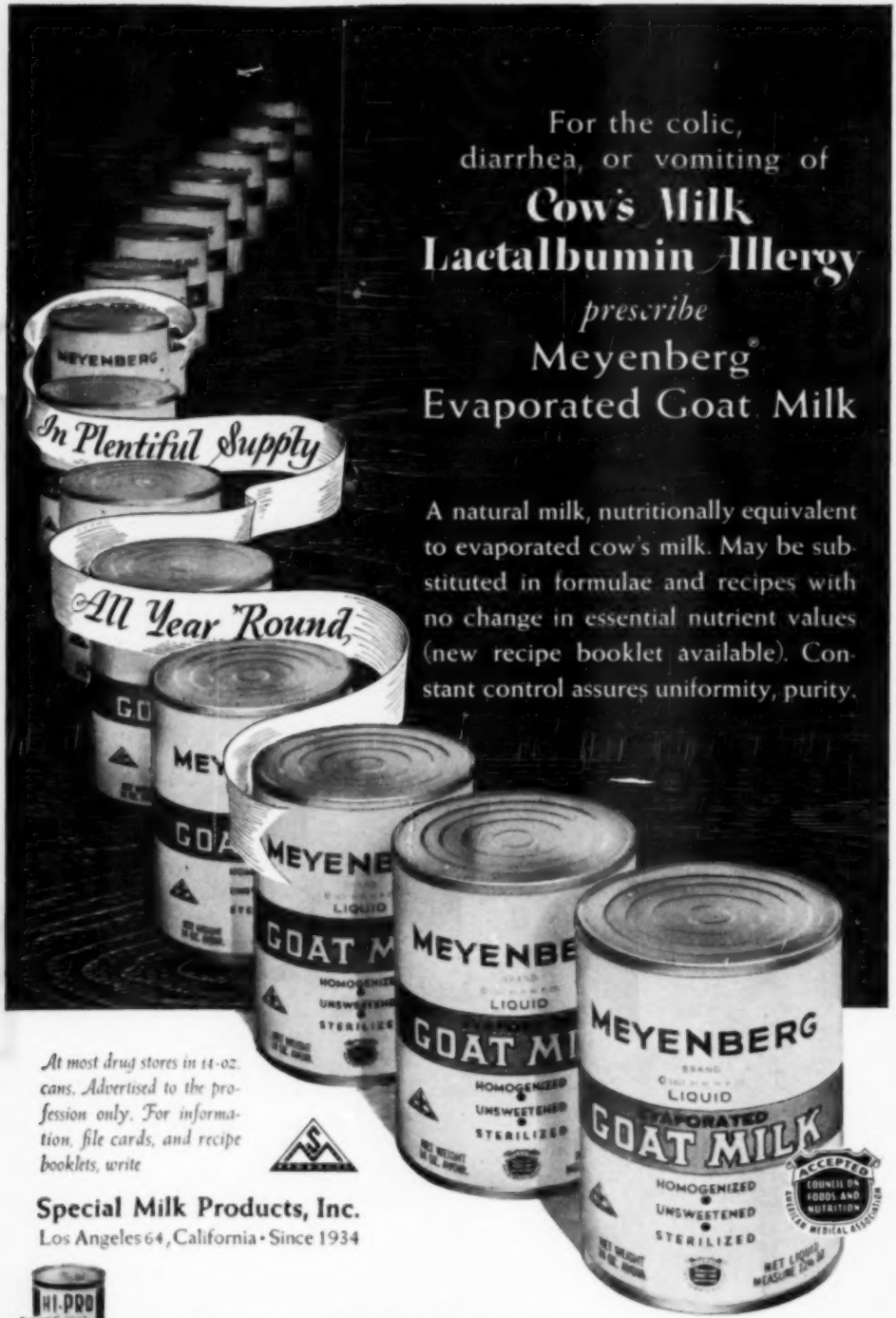
REMITTANCES should be made by check, draft, post office or express money order. If currency is sent, the letter should be registered. Stamps in amounts under one dollar are acceptable.

CHANGE OF ADDRESS NOTICE should give both the old and the new and state if change is permanent or temporary.

CONTENTS

ORIGINAL COMMUNICATIONS

(Continued on page 5)



For the colic, diarrhea, or vomiting of

Cow's Milk Lactalbumin Allergy

prescribe

Meyenberg® Evaporated Goat Milk

A natural milk, nutritionally equivalent to evaporated cow's milk. May be substituted in formulae and recipes with no change in essential nutrient values (new recipe booklet available). Constant control assures uniformity, purity.

At most drug stores in 14-oz. cans. Advertised to the profession only. For information, file cards, and recipe booklets, write

Special Milk Products, Inc.
Los Angeles 64, California • Since 1934

BY THE MAKERS OF *Hi-Pro*®—HIGH-PROTEIN, LOW-FAT POWDERED COW'S MILK.

(Continued from page 3)

CLINICAL-PATHOLOGICAL CONFERENCE

DEPARTMENT OF ABSTRACTS

ITEMS

EVERY LISTED PEDIATRIC SPECIALIST

was questioned by an independent research organization about an article in the September, 1947, issue of the Archives of Pediatrics. These specialists were asked whether they agreed with the reprint material.

Of the pediatricians who believed their experience justified an opinion, 156—81.7%—replied yes to all three points in question.

4 out of 5 Leading Pediatricians agree that CREAM OF RICE

a | **gives "more available caloric energy"** than any wheat, rye, barley, corn, oat or "baby" cereal. Of the 227 pediatricians answering (a) definitely, 192—84.6%—said yes.

b | **is "more easily digestible"** than the other cereals. Of the 248 answering (b) definitely, 212—85.5%—said yes.

c | **gives "nutritional energy more rapidly"** than the other cereals. Of the 220 answering (c) definitely, 178—80.9%—said yes.

In addition, Cream of Rice is

MOST HYPOALLERGENIC, TOO

As reported in the April, 1948, Archives of Pediatrics (pp. 183-193), by Slobody, Untracht and Hertzmark, "rice . . . shows the fewest allergic reactions of any cereal checked . . . Even children potentially allergic to rice have been shown to tolerate it well when it is cooked in the presence of moisture."

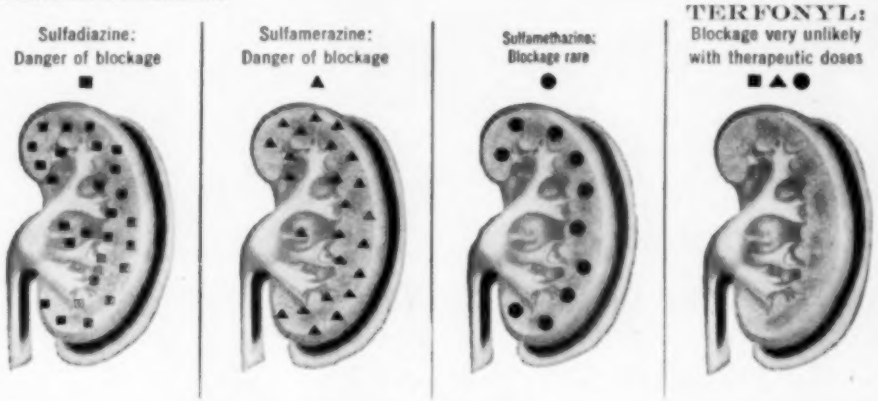
WRITE FOR PROFESSIONAL SAMPLES:
GROCERY STORE PRODUCTS CO., DEPT. AP-5,
WEST CHESTER, PA.

TERFONYL

Sulfadiazine
Sulfamerazine
Sulfamethazine

FOR SAFER SULFONAMIDE THERAPY....

Low Renal Toxicity



With usual doses of Terfonyl the danger of kidney blockage is virtually eliminated. Each of the three components is dissolved in body fluids and excreted by the kidneys *as though it were present alone.* The solubility of Terfonyl is an important safety factor.

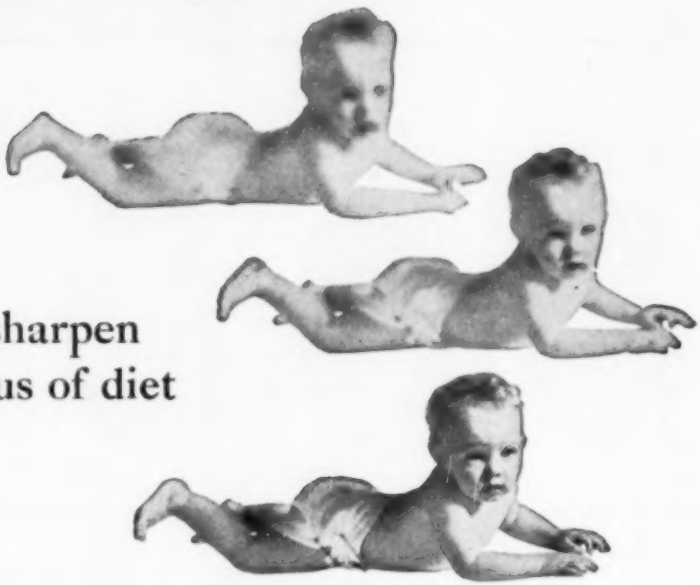
Terfonyl contains equal parts of sulfadiazine, sulfamerazine and sulfamethazine, chosen for their high effectiveness and low toxicity.

Terfonyl Tablets, 0.5 Gm. Bottles of 100 and 1000

Terfonyl Suspension, 0.5 Gm. per 5 cc.
Appetizing raspberry flavor • Pint bottles

SQUIBB MANUFACTURING CHEMISTS TO THE MEDICAL PROFESSION SINCE 1858

Flexible Formula

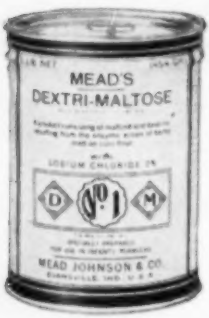


...to sharpen the focus of diet

WHEN an infant's diet is not formulated to his exact needs, it is like a picture out of focus. For an individualist, the basic formula must be *flexible* to meet the changing needs of the moment—to bring the diet "into focus."

Dextri-Maltose* has been preferred by two generations of physicians because of its exceptional flexibility in formulas using whole or evaporated milk. Quantities of this carbohydrate can be varied at will with the varying caloric requirements of the individual infant; and Dextri-Maltose is available in *five* forms to meet certain clinical conditions without disturbing the feeding routine.

Not too sweet, readily soluble and easy to use, Dextri-Maltose is highly digestible and slowly absorbed. No other carbohydrate for infant feeding enjoys so authoritative a background of clinical experience.



MEAD'S

*T. M. Reg. U. S. Pat. Off.

DEXTRI-MALTOSE

DEXTRI-MALTOSE NO. 1—with 2% sodium chloride • DEXTRI-MALTOSE NO. 2—Plain • DEXTRI-MALTOSE NO. 3—with 3% Potassium Bicarbonate • DEXTRI-MALTOSE WITH YEAST EXTRACT AND IRON • PECTIN-AGAR IN DEXTRI-MALTOSE.

Descriptive literature on request

Baby eats eagerly with no coaxing!

From the first spoonful of solid food you can recommend Beech-Nut for your young patients! That's the easy way to avoid feeding problems. No baby foods offer higher quality or finer flavor than a baby gets in Beech-Nut Cereal, Strained Foods and Junior Foods. A complete choice to meet all normal dietary needs.

Babies love them—thrive on them

Beech-Nut

FOODS for BABIES

Beech-Nut high standards of production and ALL ADVERTISING have been accepted by the Council on Foods and Nutrition of the American Medical Association.

S-M-A builds husky babies

Ready-to-use S-M-A is patterned after human milk

. . . with respect to quantity and quality of essential nutritional factors. The nutritional history of S-M-A infants is similar to that of breast-fed infants.

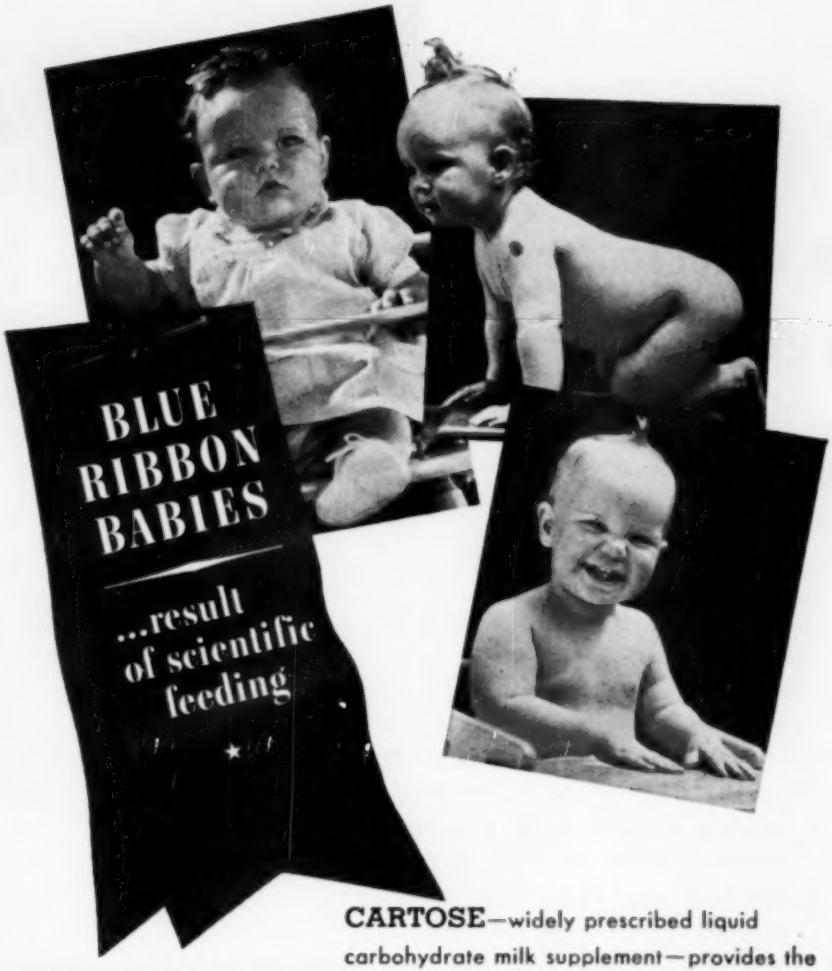
S-M-A babies are well developed, with firm tissue; they are happy and contented.

The stools of S-M-A infants closely resemble those of breast-fed infants in color, odor, consistency and bacterial flora.

VITAMIN C ADDED

S-M-A Concentrated Liquid—
cans of 14.7 fl. oz.
S-M-A Powder—1 lb. cans

Wyeth Incorporated • Philadelphia 3, Pa. *Wyeth*



CARTOSE
Mixed Carbohydrates in Easy-to-Use Liquid Form

Compatible with all milk formulas • Instantly soluble
No gumming • No nipple clogging • No caking
BOTTLES OF 1 PINT

Write for formula blanks

CARTOSE—widely prescribed liquid carbohydrate milk supplement—provides the advantage of "spaced" steady absorption in infant feeding.

Containing a carefully proportioned mixture of dextrins, maltose and dextrose—each having a different rate of assimilation—Cartose tends to minimize fermentation, colic, diarrhea or digestive disturbance.

DRISDOL®

Pure Crystalline Vitamin D_2 in Propylene Glycol • Diffuses perfectly
Tasteless... Odorless... Nonallergenic

Winthrop-Stearns INC.
NEW YORK 13, N. Y. WINDSOR, ONT.
Cartose and Drisdol, trademarks reg. U. S. & Canada

Now also milk diffusible **DRISDOL with VITAMIN A**

Life expectancy • 30 days?

While the total infant mortality has been declining, the proportion of those who died within the first month *has actually increased* from 52.7% to 62.1%*. During this fatal first month the infant should be given every possible benefit. One step in the right direction is good feeding. In this way the gastro-intestinal hazards of excessive fermentation, upset digestion and diarrhea may be minimized.

'Dexin' has proved an excellent "first carbohydrate" because of its high dextrin content. It (1) resists fermentation by the usual intestinal organisms; (2) tends to hold gas formation, distention and diarrhea to a minimum, and (3) promotes the formation of soft, flocculent, easily digested curds.

Simply prepared in hot or cold milk, 'Dexin' brand High Dextrin Carbohydrate provides well-taken and well-retained nourishment. 'Dexin' *does* make a difference.

*Vital Statistics—Special Reports: Vol. 25, No. 12, National Office of Vital Statistics, Washington, D.C. (Oct. 15) 1946, p. 206.

HIGH DEXTRIN CARBOHYDRATE **'Dexin'** BRAND

Composition—Dextrins 75% • Maltose 24% • Mineral Ash 0.25% • Moisture 0.75% • Available carbohydrate 99% • 115 calories per ounce • 6 level packed tablespoonfuls equal 1 ounce • Containers of twelve ounces and three pounds • Accepted by the Council on Foods and Nutrition, American Medical Association. 'Dexin' Reg. Trademark

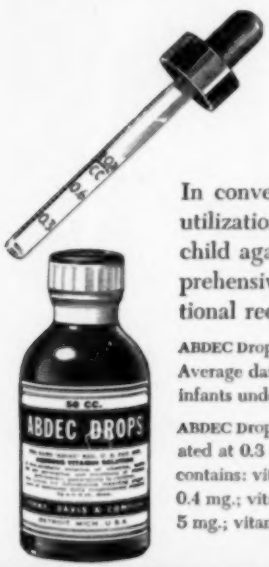
Literature on request

BURROUGHS WELLCOME & CO. (U.S.A.) INC., 9 & 11 East 41st St., New York 17, N.Y.

not "one to grow on"
but eight!

ABDEC® DROPS

8 vitamins, in convenient liquid form, for the infant and growing child



ABDEC Drops contain eight important vitamins in a stable, non-oily, non-alcoholic solution that is readily acceptable to children.

In convenient liquid form that facilitates rapid absorption and utilization, ABDEC Drops protect the healthy infant and growing child against vitamin deficiencies and afford the sick child comprehensive multivitamin therapy to meet his extraordinary nutritional requirements.

ABDEC Drops may be placed directly on the tongue or added to food, fluid or formula. Average daily dose (preferably given at a single feeding) is 0.3 cc. (5 minims) for infants under one year, and 0.6 cc. (10 minims) for older children.

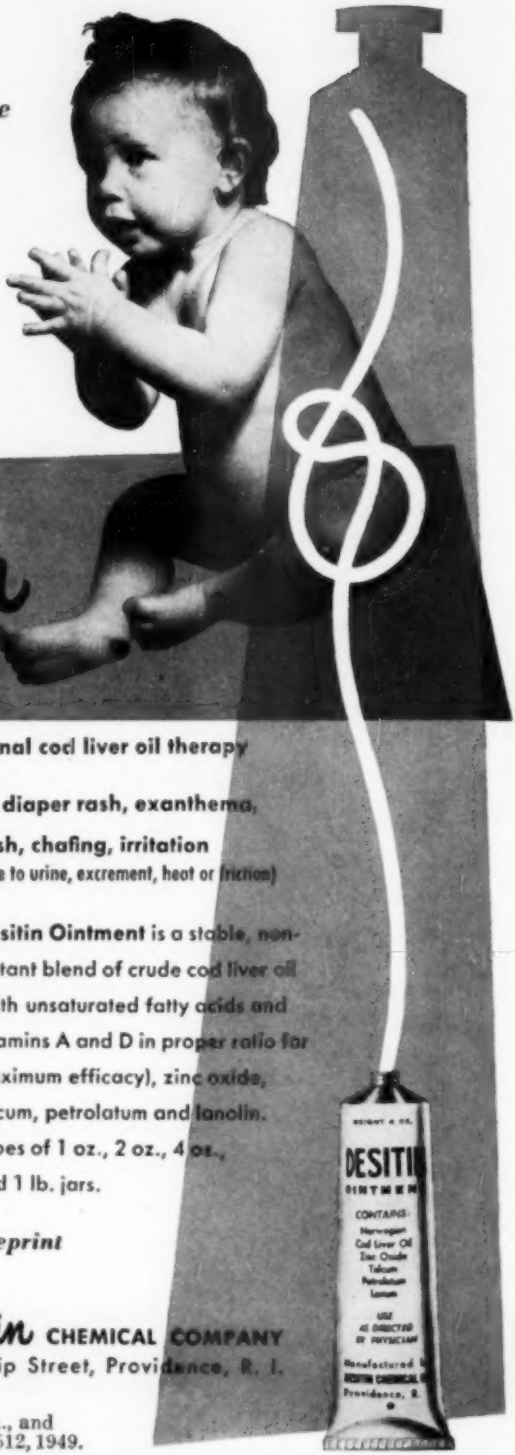
ABDEC Drops are supplied in 15 cc. and 50 cc. bottles with a special dropper, graduated at 0.3 cc. and 0.6 cc. to facilitate accurate dosage. Each 0.6 cc. (10 minims) contains: vitamin A, 5000 units; vitamin D, 1000 units; vitamin B_1, 1 mg.; vitamin B_2, 0.4 mg.; vitamin B_6, 1 mg.; pantothenic acid (as sodium salt), 2 mg.; nicotinamide, 5 mg.; vitamin C, 50 mg.

PARKE, DAVIS & COMPANY

MEDICAMENTA VERA

"This rapid healing, without exception, of the most excoriated buttocks, in so brief a time,"[1]

indicates that Desitin, the modified cod liver oil ointment, is particularly suitable for infantile intertrigo. Well established is the protective, soothing, healing influence of . . .



Desitin®

OINTMENT

the external cod liver oil therapy

in diaper rash, exanthema, rash, chafing, irritation
(due to urine, excrement, heat or friction)

Desitin Ointment is a stable, non-irritant blend of crude cod liver oil (with unsaturated fatty acids and vitamins A and D in proper ratio for maximum efficacy), zinc oxide, talcum, petrolatum and lanolin. Tubes of 1 oz., 2 oz., 4 oz., and 1 lb. jars.

℞

Send for SAMPLES and new clinical reprint

Desitin CHEMICAL COMPANY
70 Ship Street, Providence, R. I.

1. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Industrial Med. & Surg. 18:512, 1949.

ARCHIVES OF PEDIATRICS

VOL. 67 MAY 1950 No. 5

JOHN FITCH LANDON, M.D., Editor

VON GIERKE'S DISEASE

REPORT OF CASE WITH MARKED HYPERCHOLESTEROLEMIA

THEODORE M. HELLER, M.D.

AND

JOSEPH SCHWARTZMAN, M.D.

Brooklyn.

Since Von Gierke[1] reported his two cases in 1929, the total number on record does not yet exceed sixty. Because of its great rarity and because there is still much to be learned about this entity, another case of the nephrohepatic form in an infant is being reported in order to describe its unusual features.

CASE REPORT

S. H., a female infant of Jewish parentage, was born December 11, 1947, after a full term pregnancy and a normal delivery with no history of injury to the baby. The birth weight was 6 pounds 12 ounces. The baby was fed a cow's milk formula of evaporated milk, dextri-maltose and water with a supplemental ration of Vi-Fort. The infant appeared to thrive until the age of three months. A photograph taken at three months shows the patient in prone position, holding its head and trunk up firmly with its hands. At this time the patient had been ill for a few days with what appeared to be an upper respiratory infection. The infant was immunized to smallpox at about this time.

Both parents were in their early twenties, in good health, and this infant was their first child. There is no consanguinity between the parents and no history of any hereditary or familial diseases.

The patient was seven months old when first seen by the writers. The mother complained that the infant had not been gaining weight properly for four months. Although at the age of three months she had been able to support herself on her hands when prone, she no longer could do so. At the age of seven months, the infant weighed 13 pounds 4 ounces, had a head to heel length of 25½ inches. Frontal bossing was prominent. The abdomen was markedly enlarged, and this contrasted strongly with the general appearance of malnutrition. The musculature was generally flaccid. The baby remained in whatever position it was placed and appeared unable to turn from a prone to a supine position. When supine, her thighs were spread with her knees touching the underlying bed, forming a flaccid diamond. The cry was weak and whimpering and sounded like that of a younger infant. There appeared to be no definite mental retardation. The liver was markedly enlarged. It could be felt on the right side all the way down to the iliac crest. A mass felt under the left costal margin was assumed to be the spleen, but was probably the left lobe of the enlarged liver. The wrist on fluoroscopy showed the presence of two small carpal bones and corresponded closely in skeletal maturation to Todd's standard for three months.[2] The skin of the upper arms showed a number of small xanthomatous excrescences. An effort was made to improve the infant's nutrition by varying the diet, since the baby had had only diluted milk up to that time. Phenobarbital was given to control excessive regurgitation. Dessicated thyroid gr. 1/10 was given daily because of the apparent delay in skeletal maturation. Crude liver was given intramuscularly.

There followed some nutritional improvement during the next few weeks. Then the patient became acutely ill with fever which fluctuated irregularly from normal to 103° F., during which time the baby coughed and appeared to have an upper respiratory infection. Treatment with penicillin (300,000 units in oil intramuscularly daily) resulted in no apparent improvement. At this time a blood specimen obtained at home for a blood count revealed that the blood was grayish and creamy in appearance. Because of the downward trend and because of the unusual aspects of the case, the infant was admitted to the Flower Fifth Avenue Hospital

on July 27, 1948 where she expired the following day, after her temperature had risen terminally to 108° F.

Laboratory studies revealed that the blood was grossly milky with fat separating on standing. The blood hemoglobin was 120 per cent and there were 4,900,000 R.B.C. per cubic millimeter. There were 19,800 W.B.C. per cubic millimeter, with 15 per cent polymorphonuclears, all of which were adult forms, and 81 per cent lymphocytes. The urine was normal. After the administration of fluids the hemoglobin was 50 per cent and there were 2,900,000 R.B.C. per cubic millimeter. The total white count was then 52,000 per cubic millimeter with 24 per cent polymorphonuclears and 72 per cent lymphocytes.

The serum cholesterol was 2,112 milligrams per cent with 633.6 milligrams per cent esters.

The spinal fluid showed a negative Pandy reaction, 135 milligrams per cent sugar and 660 milligrams per cent chlorides. A 48-hour culture showed no growth of organisms.

An autopsy was performed by the pathology department on July 28, 1948 and the report follows.

AUTOPSY DESCRIPTION

The body is that of a white female measuring 35.2 centimeters crown-rump and 57.2 centimeters crown-heel and weighing about 5,120 grams. There is slight rigor, no lividity, slight edema of body, no cyanosis and no jaundice. The pupils are equal, regular, and measure 0.25 centimeters in diameter.

Special Marks Noted: There is marked pallor of the body. Three hemorrhagic areas are present in right upper quadrant due to diagnostic procedure. Numerous small xanthomatous lesions are present on left and right elbow and upper arm.

The subcutaneous fat varies from ¼ to ½ millimeter in thickness. The peritoneal cavity is smooth and glistening and contains a small amount of creamy red fluid. The liver margin extends seven centimeters below the costal margins in the midclavicular line. The appendix is anterior and free. The diaphragm arches to the fourth rib on the left side.

The pericardial sac is smooth and glistening and contains no excess fluid. The heart weighs 34 grams. The right chamber

appears dilated but not hypertrophied. The myocardium is pale in color but normal in consistency. The valves show nothing of note. A patent foramen ovale is present. The coronaries are thin-walled and neither narrowed nor occluded at any point. The root of the aorta is smooth and glistening.

Both pleural cavities are smooth and glistening and contain no excess fluid.

The right lung weighs 80 grams; the left, 65 grams. The trachea, larger bronchi and pulmonary vessels show nothing of note.

The parenchyma of the lung, which is pale and feathery, collapses when the thorax is opened. In the posterior part, there is hypostasis. There are scattered areas of pulmonary consolidation most marked in lower lobes suggesting bronchopneumonia. No nodules are palpable in the parenchyma. No excess fluid escapes from the cut surface and no pus can be expressed.

The spleen weighs 15 grams. The capsule is slate in color and smooth. The parenchyma is pale red and firm. The malpighian corpuscles are evident. The splenic artery and vein appear normal.

The esophagus, stomach and duodenum appear normal. The bile ducts and pancreatic ducts open together at the papilla. The rest of the intestinal tract shows no significant changes. The mesenteric and periaortic lymph nodes show nothing unusual.

The liver weighs 900 grams and is tremendously enlarged, covering the whole upper half of the abdomen. The capsule is smooth and glistening. The parenchyma is light brown in color and firm. The liver edges are sharp. The cut surface has a pale yellowish appearance, but has apparently normal lobular structures.

The gall bladder is thin-walled, filled with a moderate amount of dark colored bile, and no stones. The ducts are patent throughout.

The pancreas is of usual size, color and shape; lobulations are present throughout. The adrenals appear normal.

The right kidney weighs 61 grams; the left, 62 grams. The capsules strip easily and the surfaces remaining are smooth and glistening (except for fetal lobulations), the cortex and medulla are enlarged and the sharp demarcations between the two are absent. The kidney is pale in color. The renal pelves, the ureters and the bladder show nothing of note.

The ovaries, uterus and tubes are small but normal for an infant female.

The aorta shows no atherosclerosis. The lymph nodes, not previously described, show nothing of note.

The thyroid appears normal grossly.

The osseous system shows no significant abnormalities except for pallor.

The head was examined: Grossly the skull showed an area of deformity, the left occipital area being prominent while the right was flattened. The brain showed edema and there was an increase in fluid in the cranial cavity.

NOTE: All the organs had a creamy pale yellow appearance both grossly and on cut section. The blood also had a creamy pale red appearance suggesting lipemia.

GROSS ANATOMIC DIAGNOSIS

1. Lipemia.
2. Hepatomegaly.
3. Lipoid infiltration of liver, kidneys, bone marrow.
4. Xanthomatosis of skin of upper extremities.
5. Dilated right ventricle; patent foramen ovale.
6. Bilateral bronchopneumonia.

MICROSCOPIC DIAGNOSIS

Organ	
Adrenal	Normal
Bones	Normal
Brain	Slight edema, occasional large fat-filled macrophages.
Esophagus	Normal
Gall bladder	Normal
Heart	Extensive fatty metamorphosis.
Intestine	Normal
Kidney	Swelling of convoluted tubules, distention of tubules with glycogen granules. Fatty infiltration in connective tissue stroma. Glomeruli and collecting tubules normal.
Lung	Focal areas of bronchopneumonia, congestion.
Liver	Extensive fatty infiltration and degeneration.

	Enlargement of parenchymal liver cells with fat and glycogen.
Lymph node	Fatty infiltration with large fat-laden macrophages.
Pancreas	Normal
Spleen	Replacement of red pulp of spleen with large fat-laden macrophages.

FINAL MICROSCOPIC DIAGNOSIS

1. Von Gierke's disease (hepatorenal).
2. Lipemia and fatty infiltration of spleen, liver, heart, kidney.
3. Bronchopneumonia.

DISCUSSION

Von Gierke's disease was not diagnosed during life. The findings of apparent splenohepatomegaly (the enlargement of the left lobe of the liver was probably responsible for the clinical impression of splenomegaly), anemia and fever brought Letterer-Siwe's disease to mind. The lipemia, the yellowish skin nodules, the age, sex and Jewish ancestry of the patient made us suspect the presence of Niemann-Pick's disease. The unfortunate intervention of an upper respiratory infection, which led to bronchopneumonia and death so soon after this baby was first seen, made it impossible to carry out the indicated laboratory studies. The outstanding feature which differentiates this case from all other cases of glycogen storage disease previously reported was the marked lipemia and the exceedingly high serum cholesterol. It is evident that the xanthomatous skin lesions were secondary to the hypercholesterolemia. It has long been known that hypercholesterolemia occurs commonly in Von Gierke's disease. This was particularly stressed by Krakower[3], by Hogg and Sidbury,[4] by Van Creveld,[5] and by Beumer.[6] Nevertheless, the blood cholesterol values found by these authors generally varied from 300 to 400 milligrams per cent. Hogg and Sidbury's case showed a blood cholesterol of 420 milligrams per cent. Goodman, Shuman and Goodman[7] describe the case of a one-year-old boy with marked lipemia, xanthomatosis, hepatosplenomegaly and lipemia retinalis which closely parallels the findings which were made in our patient. Their patient improved clinically under a low fat diet. The diagnosis of spleno-

megaly was clinical only. Liver puncture was not done and the response of the blood sugar to an injection of epinephrine was not described. This case, which was considered a case of idiopathic lipemia, might well have turned out on further study to be another case of Von Gierke's disease with a similar degree of lipemia and xanthomatosis. It is notable that in their blood chemical studies these authors found that no serum lipase was present.

It may be useful at this time to take note of certain clinical observations made in previously reported cases which help us to better understand the essential nature of this disease. First of all, the rarity of this disease and the fact that there has been no established case of direct transmission from an affected parent[8] would appear to be sufficient evidence that Von Gierke's disease is not hereditary. Another interesting point is the varying age of onset. It is significant that no case has been diagnosed in the neonatal period.[8] It is clearly proved, by the demonstrable though low rise in blood sugar following epinephrine injection, that glycogenolysis does occur in Von Gierke's disease, but it takes place at a lower hypoglycemic threshold. It would seem that the glycogen which is stored is either different from normal glycogen or else that the enzymes necessary to its breakdown may not be present in their normal form or in sufficient quantity. The glycogenolytic mechanism which becomes impaired in Von Gierke's disease can do so early in infancy as it did in our case, and also in later childhood. The reports of Anderson,[9] in 1935, and that of Fliess and Bloom,[10] in 1938, indicate that it may yet be necessary to revise the notion that this disease is inevitably progressive and fatal.

SUMMARY AND CONCLUSIONS

1. A case of Von Gierke's disease in a seven-month female infant is being reported.
2. The hyperlipemia and hypercholesterolemia in this case were the highest ever reported.
3. Von Gierke's disease should be kept prominently in mind in every case of hepatomegaly in children.

The writers wish to express their indebtedness to the Department of Pathology of the New York Medical College, which performed the autopsy and prepared the report reproduced above.

REFERENCES

1. Von Gierke, E.: Beitr. z. path. Anat., 82: 497, 1929.
2. Todd, T. W.: Atlas of Skeletal Maturation, p. 134, 1937.
3. Krakower, C.: The Lipoid Factor in Glycogen Storage Disease. J. Pediat., 9: 728, 1936.
4. Hogg, P. and Sidbury, J. B.: A Case of Von Gierke's Disease with Marked Lipemia. J. Pediat., 10: 798, 1937.
5. Van Creveld, S.: Medicine, 18: pp. 119-128, 1939.
6. Beumer, H.: Neue deutsche Klinik, Ergaenzungsband, p. 336, 1933.
7. Goodman, S.; Shuman, H. and Goodman, S.: Idiopathic Lipemia with Secondary Xanthomatosis, Hepatosplenomegaly and Lipemic Retinalis. J. Pediat., 16: 596, 1940.
8. Ellis, R. W. B. and Payne, W. W.: Glycogen Disease. Quart. J. Med., 5: 31, 1936.
9. Anderson, P. M.: Glycogen Accumulation Disease. M. J. Australia, p. 362, March 23, 1935.
10. Fliess, M. M. and Bloom, S. M.: Von Gierke's Glycogen Storage Disease. Case Report with Unusual Findings. J. Pediat., 12: 635, 1938.

SEQUELAE OF MENINGOCOCCIC MENINGITIS IN CHILDREN. (Lancet, London, 2: 149, July 23, 1949). Matthews reports that among the 50 children with meningococcic meningitis treated at the Hospital for Sick Children in London over the ten year period ending in May 1948, there were 7 who died of the meningitis; 1 died eleven months later with pneumonia and bilateral mastoiditis following whooping cough. Of the remaining 42 children, 35 were seen and examined personally, 3 were followed by letter, and a doctor's report obtained where the child was not normal; 4 could not be traced. Sequelae were detected in 4 of the children, but in only 1 of them were they severe enough to necessitate admission to a special institution. This child was 13 months old when he had meningitis. He has hydrocephalus, hemiplegia and optic atrophy and is mentally defective and deaf. The other 3 children have deafness as their only disability, 2 of them being almost completely deaf in both ears while the third had bilateral otitis media simultaneously with meningitis and now has impaired hearing in the right ear. The fact that all the fatalities as well as all the serious sequels occurred in children less than 2 years old (6 of 7 deaths in children less than 1 year old) indicates that the prognosis of meningococcic meningitis is most unfavorable in the youngest age group. The most common permanent sequelae in this age group are blindness, deafness and mental defect. Comparing the death rate, as well as the incidence of sequelae in this series with those given in reports antedating the era of improved therapeutic methods, the author states that adequate chemotherapy greatly reduces the mortality. Of equal importance is the low incidence of serious sequelae.—*Journal A.M.A.*

LETTERER-SIWE'S DISEASE*

Report of Case

Howard D. Steele, M.D., C.M.
Montreal.

In 1949, two cases of Letterer-Siwe's disease were reported,[5, 7] bringing to twenty-six the number thus far recorded. This case is presented because the condition is not common, and because any knowledge, even if not entirely new, may assist in settling controversial points.

The first case was reported by Letterer[4] in 1924. The disease involves the reticulo-endothelial system, and with others, such as Gaucher's disease, Niemann-Pick's disease, eosinophilic granuloma of bone, Hand-Schuller-Christian's disease and certain xanthomatoses, is often spoken of as a reticulo-endotheliosis. It is more specifically called a non-lipid reticulo-endotheliosis to differentiate it from the lipid storage diseases of the group such as Gaucher's and Niemann-Pick's diseases.

This segregation on the basis of lipid storage is an inadequate one, and does not give an accurate picture of the disease. Gaucher's and Niemann-Pick's diseases are characterized by reticulum cells storing lipid, but in the other conditions also lipid storage is seen to some extent. Green and Farber,[1] in their description of the pathologic picture of eosinophilic granuloma, state that foam cells are not present in the lesion in the early or very late stages. Hand-Schuller-Christian's disease, regarded as a lipid storage disease, may show no evidence of lipid storage, as found by Gross and Jacox[2] in their analysis of 84 cases. In this recorded case of Letterer-Siwe's disease, some lipid storage was found, and in some other reported cases, small amounts were present in the cell cytoplasm.

An excellent recent paper by Schafer[7] presents three cases, with a review of the literature and references. Other papers, by Gross and Jacox,[2] and Jaffe and Lichtenstein,[3] review cases of Letterer-Siwe's, and Hand-Schuller-Christian's diseases, and eosinophilic granuloma of bone, and stress their inter-relationship. The latter authors, and more recently Mallory,[6] state that these diseases may be different responses to a similar basic disorder, possibly a yet unknown agent of infection. Wallgren[8] proposes that Letterer-

*From the Department of Pathology, Queens University, Kingston, Ont.

Siwe's and Hand-Schuller-Christian's diseases are different types of the same malady, the differences in clinical course and histological findings being the result of age difference and site of lesions in the two groups. While some authors are able to find a relationship to infection, in at least three cases there seems to have been none.[7]

The features of Letterer-Siwe's disease are: non-familial and non-hereditary affliction with an acute onset and fatal termination in early infancy, widespread reticulo-endothelial hyperplasia, enlargement of the spleen, liver and lymph nodes, hypochromic anemia, bone lesions and a purpuric rash. The thymus gland tissue is frequently almost completely replaced by fibrous tissue.

CASE HISTORY

At the age of 17 months, a white female child was admitted to hospital for repair of an umbilical hernia. The operation was uneventful and the child returned home. The parents noted then that the child was less active, and had a poor appetite. About one month following the operation, the parents noted swelling of the glands under the right mandible with extension behind the ear. The family doctor told the parents that a septic sore throat was the cause. The swelling persisted, and later a swelling developed on the left side of the neck. The day before her final admission to hospital, she developed abdominal distension and vomiting.

In hospital, the following past history was obtained from the parents: The child had always been anemic, a poor eater, and had frequent sore throats. Examination revealed some seborrhoeic dermatitis about the right ear. There were enlarged, rubbery lymph nodes extending from behind the right ear down along the right side of the mandible. The chest had some impaired resonance over the left base, and the child had a cough. The abdomen was distended and resonant on percussion. The spleen was enlarged 3½ fingers below the costal margin. The liver was not palpable. Some scattered purpuric spots were noted on the skin of the abdomen. The skin generally had a slight icteric tint. The blood examination revealed: Red cells, 2,670,000 per cu. mm.; white cells, 4,250 per cu. mm. with a differential count of lymphocytes 46 per cent, neutrophils 30 per cent, stab cells 20 per cent

and monocytes 4 per cent. The hemoglobin was 45 per cent and the color index 0.62. Coagulation time, 2 minutes. Sedimentation rate in the first hour was 102 mms. The platelet count was 138,000 per cu. mm. The urine was negative. An icteric index was reported as 9.8 units (normal 4-6). In hospital, the child ran a temperature of 99° to 100° F. The treatment received was routine nursing care, penicillin and a few blood transfusions.

Other white counts and differential smears were done, and the white cells ranged between 4,000 and 6,000 per cu. mm. with no appreciable differences in the smears.

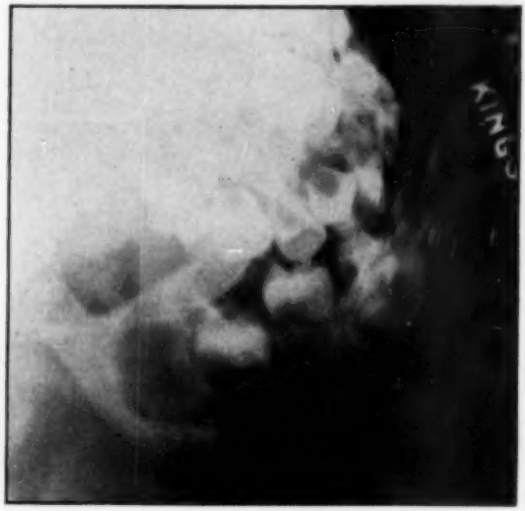


Fig. 1. X-ray of mandible to show the cystic area posterior to the molar teeth.

A blood calcium was reported as 10.5 mgm.% (normal 9-12), and the blood phosphorus 12.4 mgm.% (normal 4-7). A chest x-ray was normal, and x-ray of the abdomen showed only considerable gas in the bowel. The bones of the arms, legs, thorax and pelvis all appeared normal by x-ray, but the mandible showed cystic areas below and posterior to the molar teeth on both sides, most marked on the left. The Wassermann was negative. Three

weeks before death, the child developed a petechial rash on the trunk, neck and scalp only, most marked on the neck and upper trunk. The child died one month following admission to hospital, and about two months from the onset of symptoms, at the age of 20 months.

AUTOPSY FINDINGS

The thymus weighed 20 grams, was yellowish-grey in color, tough and rubbery in consistency, and occupied the normal site. On sectioning, much resistance was encountered, but no calcified

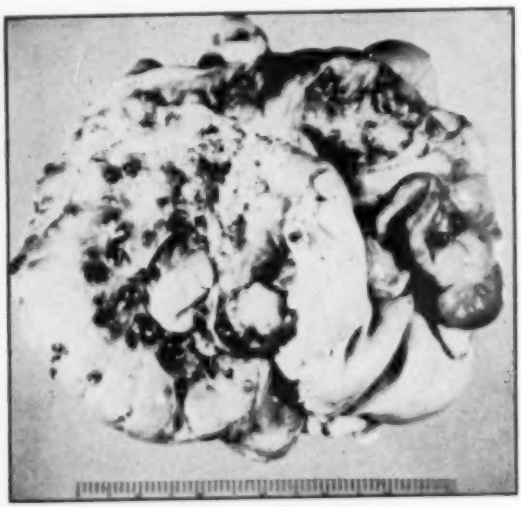


Fig. 2. Photograph of bowel to show the prominent lymph nodes in the mesentery and on the serosa.

areas were present. The cut surface was also yellowish-grey in colour, with a few darker circular areas noted.

The spleen was enlarged and weighed 250 grams. The shape was normal. On sectioning, the pulp was soft and dark red in colour. The liver was also enlarged, but appeared otherwise normal.

The lymph nodes of the neck, thorax and abdomen were all enlarged, numerous in number, dark red to blue in colour, and soft in consistency. There were numerous lymph nodes on the surface

of the bowel wall which were loosely attached and pulled away with ease. The lymphoid tissue of the bowel was also obvious by its bluish colour. This was very striking in the appendix which appeared dark reddish-blue in colour throughout. The lymph nodes of the arms and legs were not remarkable.

Attempts to remove part of the mandible were abandoned because of technical difficulties.

The remaining organs were not abnormal grossly.

MICROSCOPIC FINDINGS

The above organs which were grossly abnormal, as well as some

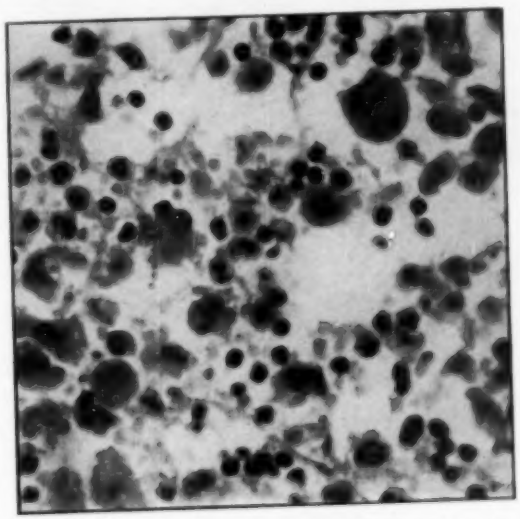


Fig. 3. Photomicrograph of lymph node, magnification 440x. These cells are typical of those found in the lymphoid tissue in this case. Some nuclear variation is evident.

others that appeared normal, showed the presence in large numbers of a large mononuclear cell which had an eosinophilic cytoplasm. Some of the cells contained two or more vesicular nuclei, with nucleoli. The lymph nodes of the neck, thorax and abdomen contained large numbers of these cells, with varying degrees of

loss of their normal architecture, and some areas of haemorrhage. The spleen also showed loss of normal structure with a widespread infiltration of the pulp by the above-mentioned large mononuclear cells, accompanied by smaller numbers of polymorphonuclear leucocytes and a few fibroblasts. The Malpighian corpuscles were largely replaced and remained only as a narrow rim of lymphocytes about the vessels. Both lungs showed large numbers of these cells, but here, some typical foam cells were present. Alveolar walls were thickened and some areas of bronchopneumonia were observed. Very small amounts of interstitial fibrosis with collagen

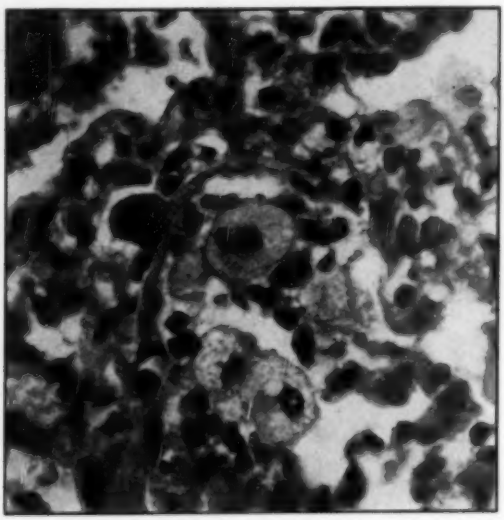


Fig. 4. Photomicrograph of lung tissue, magnification 440x. Foam cells are present as well as cells with eosinophilic cytoplasm. One of the cells contains two nuclei.

were noted scattered throughout the lung tissue. The thymus showed widespread replacement of normal tissue by fibrous connective tissue, and again, large mononuclear cells were present, some with the appearance of foam cells. Wherever these cells were present in large numbers, they were accompanied by a few polymorphonuclear leucocytes. The bone marrow did contain small

numbers of the cells noted elsewhere, but was otherwise normal. The liver showed fatty metamorphosis, but few if any of the cells in question were present.

DISCUSSION

This case presents those features previously mentioned as characteristic of Letterer-Siwe's disease. Unfortunately, the blood phosphorus was not checked. The fact that the blood calcium was normal tends to decrease the significance of this and it is therefore mentioned as an unconfirmed finding. Some authors suggest that lipid storage in a reticulo-endotheliosis is a result of the age of the lesion, hence, Letterer-Siwe's disease displays little or no storage because of its acute nature. The fibrosis is presumably also an indication of the age of the lesion. On this basis, it could be suggested that the thymus was the seat of the original lesion, and the lung, because of the foam cells and early interstitial fibrosis, was next. All other lesions were probably of later origin.

The role of infection in this case is doubtful. The child was reported to have had a septic sore throat at the time of the cervical lymph node enlargement, and a history was obtained of several sore throats prior to this. What part the surgical operation played, if any, is also unknown.

SUMMARY

1. A case of Letterer-Siwe's disease, which appears to satisfy existing criteria, is presented.

2. A septic sore throat and a surgical operation are mentioned in conjunction with the onset of the disease, but no connection is inferred between these events and the etiology or onset of the disease.

3. On the basis of the lipid present and the fibrosis, the initial or oldest lesion is thought to have been in the thymus, and the next oldest in the lungs, the remaining lesions being of shorter duration.

4. The normal serum calcium and elevated blood prosphorus are presented as unconfirmed findings.

Acknowledgements: I wish to express my appreciation to Dr. F. J. O'Connor Jr., for his permission to use this case, and to Prof. J. D. Hamilton for editing the report. The photographs were prepared by Mr. P. H. Mott.

REFERENCES

1. Green, W. T. and Farber, S.: Eosinophilic or Solitary Granuloma of Bone. J. Bone & Joint Surg., 24, 499-526, 1942.
2. Gross, P. and Jacox, H. W.: Eosinophilic Granuloma and Certain Other Reticulo-Endothelial Hyperplasias of Bone. Am. J. M. Sc., 203, 673-687, 1942.
3. Jaffe, H. L. and Lichtenstein, L.: Eosinophilic Granuloma of Bone. Arch. Path., 37, 99-118, 1944.
4. Letterer, E.: Aleukamische Retikulose. Ein Beitrag zu den proliferativen, Erkrankungen des Retikuloendothelialapparates. Frankfurt. Ztschr. f. Path., 30, 377-394, 1924.
5. Levinsky, W. J.: Non-lipid Reticulo-endotheliosis—Letterer-Siwe's Disease. Report of a Case. Arch. Path., 48, 462-474, 1949.
6. Mallory, T. B.: Medical Progress—Diseases of Bone. New England J. Med., 227, 955-960, 1942.
7. Schafer, E. L.: Non-lipid Reticulo-endotheliosis—Letterer-Siwe's Disease. A Report of Three Cases. Am. J. Path., 25, 49-84, 1949.
8. Wallgren, A.: Systemic Reticulo-endothelial Granuloma (Non-lipid reticulo-endotheliosis and Schuller-Christian's Disease). Am. J. Dis. Child., 60, 471-500, 1940.

EARLY DIAGNOSIS OF TUBERCULOUS MENINGITIS. (Deutsche Medizinische Wochenschrift, Stuttgart, 74: 1131, Sept. 23, 1949). Observations on 82 children treated for tuberculous meningitis with streptomycin at the university clinic in Göttingen in 1948 and 1949 convinced Heepe that the success of this treatment is largely determined by the time elapsing between the onset of symptoms and the streptomycin treatment. About half the patients receiving treatment before the tenth day have a prospect of cure, while of those treated after the fifteenth day only one eighth has a good prognosis. Early diagnosis and treatment are of vital importance. A gradually rising temperature, diffuse or frontal headache and vomiting are typical of the disease. The vomiting, as a rule, is not related to meals and frequently occurs in the morning. Fatigue, lack of appetite, loss of weight, restlessness, disturbed sleep, apathy, sensitivity to noises, photophobia, changes in mood and character, abdominal, joint and ear pains, sensitivity to touch, changes in bowel habits, tonoclonic convulsions, gnashing of teeth and stiffness of the neck are other symptoms. The tuberculin reaction is positive. Roentgenoscopy of the lungs may be helpful, and a spinal puncture is decisive. The puncture should not be postponed until after neck rigidity and somnolence have appeared. During the early stage, when the condition may be mistaken for influenza, typhoid, otitis, pneumonia, sepsis or gastrointestinal upsets, it is important to keep in mind the possibility of tuberculous meningitis. —*Journal A.M.A.*

PERTUSSIS IN INFANCY

A Review of 3,081 Cases

Archibald L. Hoyne, M.D.*
and
Rowine Hayes Brown, M.D.**
Chicago.

Pertussis has long been a disease of great importance in infancy and childhood. For many years it was not given the attention to which it was entitled among the acute infectious diseases, because diphtheria and scarlet fever were chiefly in the limelight. With the remarkable decline in diphtheria and the waning intensity of scarlet fever, pertussis now holds a major position among the common contagious diseases.

Our purpose in writing this paper is to demonstrate what may be accomplished in the care of pertussis patients even without resorting to specific therapy, sedatives or other forms of medication which are generally used. The prominent position held by whooping cough is indicated by some of the figures which follow.

In 1934, 265,269 cases of whooping cough were reported in the United States.[1] Actually, the figure must have been far greater, since it is known that in some states a large percentage of whooping cough is not reported. Furthermore, there is no doubt that many deaths attributed to bronchopneumonia occur in infants or children who have this condition as a complication of pertussis which is never reported. In the same year (1934) there were 220,050 cases of scarlet fever, and 41,145 of diphtheria, according to the United States Public Health Service.[1] Neither of these diseases approximated whooping cough in regard to prevalence, and the number of deaths attributed to pertussis was 7,158, whereas for scarlet fever it was only 2,443, and for diphtheria, 4,218.

By 1947 there was a remarkable decline for most of the acute infectious diseases, but whooping cough continued to hold its place of high incidence. The United States Public Health Reports[2] disclose that during 1947 there were 155,991 cases of whooping cough, 84,379 of scarlet fever and 12,405 of diphtheria. This would indicate that within a period of 14 years the morbidity of

From Municipal Contagious Disease Hospital, Chicago Health Department.
*Formerly Medical Superintendent.
**Formerly Assistant Medical Superintendent.

whooping cough had receded 41 per cent, whereas scarlet fever had declined 61.6 per cent, and diphtheria 71.2 per cent. The decline in diphtheria may be readily understood because of the possibilities which active immunization affords for elimination of this disease. However, the recession for scarlet fever cannot be explained in a similar manner. In addition, it is not likely that immunization has been carried out on a sufficiently broad plane to account for the reduction of pertussis. Nevertheless, regardless of what the reason may be for the lower incidence of these diseases, whooping cough still maintains its place of eminence above scarlet fever and diphtheria. Furthermore, the 1947 mortality figures in the United States for these three diseases were respectively, 1,954, 107, and 799.[3]

It is true that the comparison in regard to whooping cough prevalence for the two years cited may not be accurate, for the reason that less attention was centered on pertussis in 1934 than in 1947. Consequently, a smaller proportion of cases was probably reported in the former year than in the latter. If this is true, it could explain why the actual recession did not appear to be still greater.

The status of whooping cough in Chicago is probably more favorable than in the nation as a whole. In 1934 there were 6,772 reported cases with 64 deaths, according to the Chicago Health Department. In the same year, there were 10,032 cases of scarlet fever with 127 deaths, and 327 of diphtheria with 41 deaths. But in 1947 the number of pertussis cases reported in the same city was 1,773 with 6 deaths, whereas there were 1,412 cases of scarlet fever with no deaths, and only 26 cases of diphtheria with 3 deaths. The foregoing statistics are presented for the purpose of emphasizing the consideration which pertussis deserves.

On the basis of the figures set forth for Chicago, we found it interesting to note when comparing the year 1947 with 1934, that the prevalence of both scarlet fever and diphtheria had each declined approximately 90 per cent, the exact figures being 85.8 per cent for the former and 92 per cent for the latter. On the other hand, the recession for whooping cough was but 73 per cent. Another interesting comparison is the reduction in the number of deaths for each of the diseases referred to. Although deaths from pertussis declined 90 per cent, scarlet fever deaths were diminished

100 per cent, and the number of deaths from diphtheria receded 92.6 per cent. We feel that both the incidence for whooping cough and the number of deaths have been influenced, like many of the other acute infectious diseases, by the higher scale of living that prevails, and which contributes generally to an improved state of nutrition, and increased resistance to infection. Nutrition, sanitation, including good ventilation, are all of vital importance in relation to whooping cough, and undoubtedly affect both morbidity and mortality rates.

Unlike many of the acute infectious diseases, the infant is susceptible to whooping cough any time after birth. Furthermore, pertussis is always serious when contracted during the first year of life. The prognosis is particularly unfavorable in infants under six months of age, and most of the fatalities occur during the first twelve months. For example, in 1947 among 1,954 pertussis deaths in the United States, 1,481, or 75.7 per cent, were less than one year of age. It is for those reasons that the accomplishment of active immunization, as originally recommended after the first half year of life, obviously could not reduce the mortality figures for infants less than six months old. Moreover, when immunization is undertaken during the second half year of life, generally little or no protection is afforded a child prior to 12 months of age. Even now, when immunization procedures are instituted at three months, or earlier, there is still a lapse after birth during which the infant is susceptible. This demonstrates again that in spite of the most approved methods for prevention, whooping cough may occur during the first few weeks if infants are exposed to the disease. Sometimes immunization of the mother during pregnancy seems to afford protection to the infant at birth.

Among our entire 3,081 patients we were able to secure a history of the administration of prophylactic vaccines in only 68 instances. Therefore, because it is well recognized that pertussis may be contracted before immunization can be accomplished, attention is often concentrated on measures of therapy thought to be appropriate. In the past a great variety of drugs and vaccines has been recommended for the treatment of whooping cough. Only within comparatively recent years has hyperimmune serum been used and accepted as an effective agent. During a still briefer period, sulfonamides have been resorted to, and now we have in

addition both penicillin and streptomycin, and also aureomycin and chloromycetin. Nor have sedatives been ignored. The hunt for an effective and simple therapeutic remedy to cure whooping cough has been conducted for many years. However, while resorting to drugs efficient measures have frequently been neglected. We believe that this fact is demonstrated by the results obtained according to the plan of treatment which was adhered to for nearly all our patients. Furthermore, our results tend to support the view that the general decline in morbidity and mortality from whooping cough in recent years may be explained by the higher plane of living to which the nation has become accustomed.

Until 1934 whooping cough patients were scarcely ever admitted to Municipal Contagious Disease Hospital. The reason for this was that the bed capacity was usually exhausted by diphtheria and scarlet fever patients. During 1934 and 1935 the total number of whooping cough infants admitted was only 17. But in 1936, an entire hospital floor was assigned for pertussis patients, and in that year there were 132 admissions. The building in which the whooping cough patients are housed is constructed according to the cubicle plan. There are 12 cubicles extending along each side of the building for almost its entire length. Each cubicle is 12 feet in depth and 10 feet wide. All partitions are of plate glass and 6 feet 8 inches in height. The ends of the cubicles are also walled off with glass partitions leaving only a doorway for entrance, with a large window in the outside wall of the cubicle. This arrangement provides for a type of ventilation which ordinarily is not possible in a completely walled ward. Through the center of the hospital floor is a glass-enclosed corridor for visitors. Entry to this corridor is made possible by an elevator which is accessible from the basement and emits passengers only into the visitors' aisle. By this arrangement parents may see their children, but there is no possible contact between visitors and the patients. Each cubicle will accommodate two patients, and for each there is a bedside table and all individual utensils which are required. There is also a large hopper with hot and cold running water in every cubicle. There are two disposal containers, one for diapers, the other for paper towels. And in addition, a laundry bag, supported by a metal stand in such a manner that it is possible to deposit soiled linen without touching the container. Individual

gowns hang near the entry to each cubicle, and the personnel is required to wear face masks for the protection of the patients. The entire technique is conducted according to medical aseptic measures. At one end of the ward is a large room equipped with oxygen tents, and here the critically ill infants are cared for. There is also a small treatment room where minor surgical work can be carried out. In addition, there is a linen room, and dressing room for nurses. In the latter, all nurses change their outer clothing for ward garments before assuming their duties. There is also a kitchenette with a spacious refrigerator, and a steam sterilizer for cups and similar utensils. Gowns and face masks are donned by physicians outside the ward, in preparation for attending the patients. All feedings are prepared in a formula room in the basement.

PLAN FOR ADMISSION OF PATIENTS

For a number of years only whooping cough patients under one year of age have been admitted to Municipal Contagious Disease Hospital. They are accepted for treatment regardless of whether complications are present or not. In fact, it is the policy of the Health Department to encourage parents to send the little patients to the hospital if there is no family physician in attendance, or if the family physician recommends such action. This is done particularly where home conditions are poor. Even when attacks are not extremely severe, if the home conditions are not good, the patient is much more likely to develop complications than if treated in the hospital. Consequently, all our patients are not admitted with complications.

In the hospital receiving room each child receives a thorough physical examination, and is vaccinated against smallpox if no vaccination scar is present. This latter procedure is omitted only during intensely warm seasons of the year, or if the patient is suffering from eczema. Nose and throat cultures for both diphtheria bacilli and hemolytic streptococci are obtained, as well as a cough plate for H. pertussis. In addition, a white blood count with differential is done, and a hemoglobin reading is made. If the infant has any skin condition, or is suspected of having another acute infectious disease, or believed to have no contagious condition at all, it is placed in a cubicle alone. When available cribs

TABLE I

3081 PERTUSSIS PATIENTS

YEARS	SEX		RACE			AGE		DEATHS		FATAL PER CENT		TOTAL DEATHS	FATAL. RATES
	M.	F.	W.	B.	MS.	UNDER 6 MOS.	6 MOS.-1 YEAR	UNDER 6 MOS.	6 MOS.-1 YEAR	UNDER 6 MOS.	6 MOS.-1 YEAR		
1934-1938	358	355	468	231	14	407	306	54	32	13.2	10.4	86	12.0
1939-1943	577	617	792	382	20	695	499	39	15	5.6	3.0	54	4.5
1944-1948	601	573	733	525	13	816	358	19	10	2.3	1.6	29	2.4
TOTALS	1536	1545	1993	1138	47	1918	1163	112	57	5.8	4.9	169	5.4

permit, every new patient is assigned to a cubicle where it remains for at least a week before placed in a cubicle with another whooping cough patient.

During the 15-year-period, from 1934 to 1948, inclusive, there were hospitalized 3,081 whooping cough patients under one year of age. In Table I, our patients are divided chronologically into three groups of five years each. For the different periods patients are classified according to age, sex and race. There were also three Japanese who are grouped according to age and sex, but do not appear in a separate column under race in our table. The number of deaths are also shown, and the fatality rates for those who were six months and less, and for those in the second half year of life. It is interesting to note, in regard to numbers that there is no significant difference in the sexes. In fact, among the total 3,081 patients there was an almost equal distribution. In the past we have sometimes thought that Negro infants were particularly susceptible to whooping cough, but our hospital figures would suggest exactly the opposite. In respect to age, it is noteworthy that 62.2 per cent of the total patients were in the first half year of life, which means that the number was 39.9 per cent greater than for those between the ages of six months and one year. Patients were also classified according to the degree of their illness as mild, moderate or severe. These groups numbered 899, 1,535 and 647, respectively.

COMPLICATIONS

In Table II, the principal complications are listed according to age, for each of the three five year periods. The total number was 2,909. Although we mentioned previously that some of the patients were admitted without complications, it is evident from the figures that many complications occurred, but most were present at the time of admission or developed soon after. Exclusive of cyanosis, which is classed as a complication, bronchopneumonia, as might be expected, was the commonest source of danger, and was present in nearly one-fourth (23.7 per cent) of the patients. Two hundred and fifty of the infants, or 8.1 per cent, had convulsions; 240, or 7.7 per cent, suffered from catarrhal otitis media, and 103 additional patients, or 3.3 per cent, had purulent discharge from one or both ears. In 226 instances, or 7.3 per cent, umbilical

TABLE II

PRINCIPAL COMPLICATIONS - 3081 PERTUSSIS PATIENTS

	1934 - 1938 AGE		1939 - 1943 AGE		1944 - 1948 AGE		TOTAL AGE		GRAND TOTAL
	0-6 Mo.	7-12 Mo.	0-6 Mo.	7-12 Mo.	0-6 Mo.	7-12 Mo.	0-6 Mo.	7-12 Mo.	
Bronchopneumonia	87	80	138	119	216	92	441	291	732
Cyanosis	154	94	186	120	286	85	626	299	925
Convulsions	38	36	56	46	55	19	149	101	250
Encephalitis	19	14	2	5	5	1	26	20	46
Umbilical Hernia	20	13	60	23	95	15	175	51	226
Catarrhal Otitis	33	30	46	55	39	37	118	122	240
Purulent Otitis	20	19	24	28	8	4	52	51	103
Conjunctivitis	6	2	11		17	8	34	10	44
Conjunctival Hemorrhage	2		6	2	2		10	2	12
Diarrhea	6		6	1	10	4	26	5	31
Thrush			7	2	4	2	11	4	15
Furunculosis			7	2	2	1	9	3	12

hernia developed as a result of the severity of the paroxysms. Various other complications of lesser frequency are also noted.

TREATMENT

For patients admitted without complications, our chief reliance was placed on the excellent facilities afforded for good ventilation, proper feeding and superior nursing. In general, no sedatives were given for the control of paroxysms, and, as a rule, special medication of any kind was omitted. If a paroxysm followed a feeding, and terminated with emesis, 15 minutes were allowed to elapse and then a second feeding was provided. Strict attention was given to the frequent changing of diapers, and to this fact we attribute the almost total absence of excoriations and skin infections. During the entire 15 years which are the subject of this report, there has never been an outbreak of diarrhea or impetigo.

Although few drugs were used, there were 59 cases among our total series who received hyperimmune serum, either in unconcentrated or concentrated form. In the concentrated form, we have believed that even as little as a single dose of 2.5 cc. injected intramuscularly often seemed helpful. However, in some instances this same dosage was repeated a second or third time. All infants, who were cyanotic or had convulsions, were placed in an oxygen tent with the least possible delay. Likewise, patients with bronchopneumonia received similar treatment. Patients requiring the services of an oxygen tent were cared for in one large room which has been previously mentioned. Pneumonia patients were given penicillin, one of the sulfonamides, or both, as the occasion demanded. In many instances we found that blood transfusions in amounts of from 50 to 150 cc. were helpful. In other cases, 5 to 10 per cent glucose was beneficial. Streptomycin, which has received considerable attention as a valuable therapeutic aid for pertussis, has been used by us to a limited degree only. Because of the small number of patients treated with streptomycin, we do not feel justified in expressing a definite opinion concerning its effectiveness. Nevertheless, thus far it has not impressed us as being remarkably helpful. Since this report was first drafted we have treated a number of patients with aureomycin and considered it much more effective than streptomycin. Chloromycetin seems even better.

Among the total 3,081 patients, 17.4 per cent were given a sulfonamide and only 9.8 per cent received penicillin, but 23.8 per cent were treated with oxygen. The average number of days ill at the time of admission was 10.6, and the average duration of hospital stay was 27 days.

Kohn and Fischer,[4] in reporting the treatment and management of 887 infants ill with whooping cough, adopted procedures very similar to our own. Their fatality rate for 887 patients less than one year of age was 4.9 per cent, an excellent figure notwithstanding the chief reliance for therapy did not rest on various drugs and sera. During the same period of time we were still more fortunate using similar measures for treatment, since among 1,402 patients less than one year of age, our fatality rate was 3.2 per cent. These facts emphasize the value of our contention that various drugs are not the primary requisites in the treatment of whooping cough.

Moreover, Place, Keller and Shaw,[5] in a study of serotherapy in pertussis, divided 150 consecutive hospital patients into four groups, which were treated in the following manner: One was given horse serum, one hyperimmune rabbit serum, another human hyperimmune serum, and 48 controls comprised the last group. There were no deaths. The authors report that no significant differences were evident in the clinical course of any of the patients.

Among our patients, in the first five years there were 713 cases with 86 deaths, or a fatality rate of 12 per cent; during the next five years (1939-1943) the total was 1,194 with 54 deaths, or a fatality rate of 4.5 per cent; for the third five years (1944-1948) admissions totalled 1,174 with 29 deaths, or a fatality rate of 2.4 per cent. Only 2 deaths occurred in 1947 among 270 patients, or a fatality rate of 0.74 per cent. Moreover, it should be mentioned that in the following year (1948), among 217 patients there were only 3 deaths, or a fatality rate of only 1.3 per cent. For the entire series of 3,081 patients under one year of age, the fatality rate was 5.4 per cent. Among the total 169 deaths, thirty patients died within 48 hours of admission; most of these 30 terminated fatally within 24 hours after entering the hospital. Exclusive of the 48 hour deaths, the fatality rate for 3,051 patients was 4.5 per cent.

SUMMARY

Three thousand eighty-one infants, under one year of age, with pertussis were hospitalized. One thousand nine hundred eighteen, or 62.2 per cent, were less than six months of age. One thousand eight hundred ninety-three (61.1 per cent) were white and 1,138 (36.9 per cent) were Negroes, and 47 (1.2 per cent) were Mexican. There were also 3 Japanese. Fatalities for the principal groups were: White, 114 (6.0 per cent); Negro, 51 (4.4 per cent); Mexican, 4 (8.5 per cent). Among the 1,918 under six months of age the fatality rate was 5.8 per cent, and for the 1,163 in the second half year of life, the fatality rate was 4.9 per cent. In respect to distribution, there was no significant difference in the sexes, and the fatality rates were approximately the same, being for the males 5.6 and for the females 5.3 per cent. The fatality rate for the entire 3,081 patients was 5.4 per cent. However, it is interesting to note that for 270 patients treated in 1947 the fatality rate was less than one per cent (0.74 per cent), and for the year 1948 the corresponding figure for 217 patients was only 1.3 per cent. For treatment, chief dependence was placed on good ventilation, proper feeding and efficient nursing. Next in order we consider oxygen, hyperimmune serum and blood transfusions. Pulmonary complications were treated with either a sulfonamide or penicillin, or both. Sedatives were seldom resorted to.

CONCLUSION

We believe the primary requisites for the treatment of pertussis are good ventilation, proper feeding and superior nursing.

Note: The year 1947 is used for comparison because later figures for mortality throughout the United States were not available.

BIBLIOGRAPHY

1. U. S. Treasury Department, Public Health Service: The Notifiable Diseases, Prevalence in States, 1934: Supplement to No. 117.
2. Public Health Reports: Notifiable Diseases, Year 1947: Vol. 63, No. 12, March 19, 1948.
3. Vital Statistics Special Reports: National Summaries: Deaths and Death Rates for Selected Causes by Age, Race and Sex, United States, 1947. Vol. 31: No. 11, Sept. 19, 1949.
4. Kohn, J. L. and Fischer, A. E.: Management of Whooping Cough with Special Reference to Infants. Am. Jour. Dis. Child. 73: 663, June 1947.
5. Place, E. H.; Keller, M. J. and Shaw, E. W.: Serotherapy in Pertussis. J. Pediat., 34: 669, June 1949.

428 *Oakdale Avenue.*

TYPHOID FEVER IN CHILDREN*

ROLAND B. SCOTT, M.D.
L. OTTO BANKS, M.D.
AND
ROBERT P. CRAWFORD, M.D.
Washington, D. C.

Recent advances in water and food sanitation, public health education and vaccination have caused a definite decline in the morbidity and mortality of typhoid fever in the United States. The true incidence and death rate of typhoid fever are actually not known because of inadequate reporting of the disease and the tendency to report typhoid and paratyphoid fevers as a single disease. The incidence[1] for typhoid and paratyphoid fevers has dropped from 160 cases per 1,000,000 population in 1934-35 to 39 in 1944-45, whereas the death rate[2] from these diseases has decreased from 1.5 per 100,000 population in 1934 to 0.3 in 1947. This decline in incidence, together with the atypical picture which typhoid fever may present in small children, favors the possibility that some cases may be overlooked. The disease still represents a public health menace in some rural areas as well as in urban communities where the carrier state may play an important role in the epidemiology.

Typhoid fever is less common in infants and small children than in adults. The symptomatology is more variable in children but in general the course is shorter. However, the mortality tends to be higher in the very young.[3]

We desire to report clinical observations on 18 cases of typhoid fever in Negro children admitted to the pediatric service during the years 1939-49. Ten of the patients were boys and the remainder were girls. Seven of the children lived in Maryland, six in Virginia and five in the District of Columbia. One-third of the reported cases were admitted in 1943 whereas the other 12 cases were about evenly distributed over a nine-year period. The increased incidence for 1943 was attributed to the inauguration of a trailer camp unit in nearby Virginia to house an influx of war workers.

That forty-nine per cent of the cases were admitted in the winter

*From the Department of Pediatrics, Howard University, School of Medicine, and the Pediatric Service, Freedmen's Hospital, Washington, D. C.

season is not in keeping with the autumnal incidence usually reported for this disease (see Table 1).

TABLE 1

Month	Cases	Month	Cases
January	6	September	1
February	1	October	2
March	1	November	1
July	3	December	2
August	1		

The distribution of cases according to age is tabulated in Table 2.

TABLE 2

Age Range	Cases	Age Range	Cases
0-2 yrs.	3	5-10 yrs.	7
2-5 yrs.	3	10-12 yrs.	5

The youngest patient was 4 months, the oldest 12 years (the maximum age admitted to our service) and the average age was 5.5 years.

COMPLAINTS AND DIAGNOSES ON ADMISSION

In only six of the cases was the diagnosis of typhoid fever made on admission, although in two additional cases the condition was included in the differential diagnosis. The other cases were admitted as pneumonia (3), meningitis (2), diarrhea (1), mesenteric adenitis (1), rheumatic fever (1), diabetic acidosis (1) and undetermined (1). The most frequent admitting complaints are listed in Table 3.

TABLE 3

Complaints	Cases	Complaints	Cases
Abdominal pain	8	Anorexia	4
Diarrhea	6	Headache	4
Fever	6		

SYMPTOMATOLOGY

During hospitalization, protean signs and symptoms were noted as revealed in Table 4.

TABLE 4

Symptoms	Cases	Symptoms	Cases
Fever	18	Bronchitis	6
Anorexia	17	Cough	5
Drowsiness	16	Stiff Neck	4
Abdominal pain	14	Melena	4
Tonsillitis	12	Constipation	2
Diarrhea	11	Convulsions	2
Vomiting	10	Enlarged spleen	2
Irritability	9	Epistaxis	2
Dyspnea	7	Rose spots	1
Delirium	6		

The lowest temperature on admission was 99° F., the highest 104.6° F. and the average for the cases was 102.9° F. The fever curves were continuous in 8 cases and irregular in the remaining 10 cases. The shortest duration of fever was 5 days, the longest 77 days and the average 22.8 days.

Over 80 per cent of the children were lethargic. Abdominal pain was a very frequent symptom. Respiratory complaints (tonsillitis, dyspnea, bronchitis) were common findings and occasionally accounted for errors in diagnosis on admission. Symptoms of central nervous system involvement (delirium, convulsions and stiff neck) were present in over half of the patients and two cases were admitted with the diagnosis of possible meningitis. Enlarged spleen and rose spots were rarely encountered. The average pulse was 111.2 per minute and the dicrotic character was not encountered in any case.

LABORATORY DATA

Thirteen cases had positive blood cultures during the first week. Six cases had positive stool cultures while only four patients had positive urine cultures. Positive stool or urine cultures in conjunction with a suggestive history and diagnostic titer with the "O" antigen in the Widal test were considered as evidence of active infection.

A diagnostic agglutination reaction (1:80 or above)[4] was obtained in 16 cases. In four cases the organism was not recovered in cultures; however, the diagnosis was established on the basis of a suggestive history, physical findings and unusually high agglutination reactions for the "O" antigen.

The white cell count was often an early clue to the correct diagnosis. The highest count was 17,800, the lowest 3,400 and the average 7,165 per cu. mm. Fifteen cases (83 per cent) showed a definite leukopenia. Lymphocytosis was found in 10 cases. An accompanying mild anemia was found in most cases, the average red cell count being 3.81 million.

SOURCE OF INFECTION

The source of infection was definitely traced in six cases. Carriers were found in the families of two patients. Four children contracted the disease at a trailer camp. The source of infection in 12 cases was vague; however, ten of these admitted drinking well water.

COMPLICATIONS

The five patients in this series who developed significant complications exhibited subcutaneous abscesses (2 cases) and intestinal perforation with peritonitis, parotitis and otitis media (one case, respectively).

All of the children recovered except two. Intestinal perforation and generalized peritonitis was the primary cause of death in one case. Malnutrition, debilitation and otitis media were contributing factors in the death of a four-month-old infant.

TREATMENT

The active therapy in nine cases observed during the period 1939 to 1943 was supportive with the administration of high caloric diets, parenteral fluids and antipyretic measures. The average duration of fever in these cases was 23.3 days and the hospitalization stay averaged 44.2 days. All of these patients recovered. Subsequently, all patients received these supportive measures in addition to chemotherapy and antibiotic agents.

Sulfadiazine was used in three cases, sulfathiazole, polymyxin, aureomycin and streptomycin, respectively, in one case each with no improvement in the clinical condition or fall in the temperature. The four-month-old debilitated infant with otitis media, who received sulfathiazole, died. The average duration of the fever in cases treated with the above drugs was 34.8 days and the hospitalization period averaged 47.6 days.

Chloromycetin was the most successful therapeutic agent in our hands. Three patients received this drug. The average duration of fever was 6.3 days and the average hospitalization was 26.3 days. These cases, two of whom recovered, are reported briefly.

CASE REPORTS

Case 1. J. A. R., a 11-year-old Negro male, from Midland, Virginia, was admitted on July 20, 1949. He had been ill for three weeks and complained of abdominal pain, fever and intermittent diarrhea. He had received penicillin and sulfa drugs from a local physician for one week without improvement in his condition. Drinking water was obtained from a well. Examination revealed a severely malnourished and dehydrated child with a temperature of 101° F. Generalized abdominal tenderness and muscular splinting were noted. A plain film of the abdomen revealed air under the right leaf of the diaphragm. Admission white cell count was 8,000 with a differential as follows: neutrophiles 20, lymphocytes 75, monocytes 4 and eosinophiles 1 per cent, respectively. The icterus index was 25 units. Blood culture on July 20, 1949, revealed Eberthella typhosa which gave a positive reaction in the dilution of 1:1280 against known antityphoid serum. Stool and urine cultures were sterile and the Widal reaction was negative. Generalized abdominal distention, rigidity and tenderness were noted on July 21, 1949. Intestinal decompression was instituted. Chloromycetin was started on July 22, 1949, in a dosage of 750 mgs. every three hours by rectum, but the next day the drug was given through a Levine tube. Parenteral fluids were administered daily. The course was rapidly downhill and death occurred on July 27, 1949.

Necropsy revealed (1) perforation of the distal ileum with formation of localized pelvic abscess and generalized fibrinous peritonitis and (2) ulcerative enterocolitis with hyperplasia of the spleen and mesenteric lymph nodes.

Case 2. D.R.H., a 10-year-old Negro male, from Bowie, Maryland, was admitted on September 30, 1949, with a five-day history of fever, malaise, anorexia and somnolence. Drinking water was obtained from a well. At the time of entry the temperature was 103° F. and there was generalized abdominal tenderness. The white cell count on admission was 6,800 with a differential as

follows: neutrophiles 49, lymphocytes 45, monocytes 5 and eosinophile 1 per cent, respectively. Blood and stool cultures were positive for Eberthella typhosa. The Widal reaction on October 10, 1949, was positive in the dilution of 1:640 for the "O" antigen. The therapy consisted initially of supportive measures and penicillin. On the fourth day, when the organism was recovered, chloromycetin was started in the dosage of one gram stat and 500 mgs. every four hours. The drug was continued for 14 days after the fever returned to normal. The child appeared more alert, active and his appetite increased after 24 hours of chloromycetin therapy. He became afebrile in 16 hours and made an uneventful recovery. Blood and stool cultures remained sterile. One positive urine culture for E. typhosa was obtained on October 25, 1949, but repeated cultures subsequently were negative.

Case 3. C. R. S., a 10-year-old male, from Pisgah, Maryland, was admitted on October 5, 1949 with the complaints of epigastric pain, fever, nausea and diarrhea of 10 days duration. Water was consumed from a well situated at the foot of a hill on which the privy was located. The mother and sister gave a history of typhoid fever many years previously. Examination revealed a temperature of 101° F., sordes on the tongue and an injected pharynx. The white blood cell count on admission was 7,400 with the differential of neutrophiles 65 and lymphocytes 35 per cent, respectively. Blood and stool cultures on admission were positive for Eberthella typhosa. Urine cultures were consistently sterile. The patient was started on supportive measures and penicillin which were continued for five days. Chloromycetin was started on the fifth day in the dosage of one gram initially and 500 mgs. every four hours. It was continued for 14 days after the fever returned to normal. The temperature fell from 102° to 99° F. after 20 hours of chloromycetin therapy. Cultures of the blood, feces and urine remained sterile. Recovery was prompt and uneventful.

SUMMARY

Eighteen cases of typhoid fever in Negro children have been reviewed. There were 10 boys and 8 girls in this series. Sixty-six per cent of the cases were in the age range of 5-12 years. Fever, anorexia, drowsiness, abdominal pain and tonsillitis were the most frequent symptoms observed in these cases. Fourteen patients had

positive blood, urine or stool cultures. Five patients had complications, one serious enough to cause death. From 1939-43 therapy in nine cases was mainly supportive. The remaining nine cases received in addition to supportive measures chemotherapeutic and antibiotic agents. Sulfonamides, penicillin, streptomycin, aureomycin and polymyxin were used with no improvement in the clinical condition or significant fall in the temperature.

Chloromycetin was used in three cases. Excellent results were obtained in the two cases that recovered. The value of the drug in the third case was difficult to appraise because of the serious complication (intestinal perforation and generalized peritonitis) which caused death. The mortality in this series was 11.1 per cent.

The blood culture is of paramount value in establishing the diagnosis of typhoid fever in infants and small children who present unexplained fevers.

The authors wish to acknowledge the assistance of Dr. Sidney Ross in obtaining the chloromycetin employed in the therapy of three of these cases.

BIBLIOGRAPHY

1. Metropolitan Statistical Bulletin, 27: 9, June 1946.
2. Metropolitan Statistical Bulletin, 26: 4, Jan. 1945.
3. Brennemann's Practice of Pediatrics, Vol. II, Chap. 30, p. 7.
4. Levinson, S. and MacFate, R.: Clinical Laboratory Diagnosis, 3rd Ed., p. 538.
5. Blackader, A. D.: ARCH. PEDIAT., 64: 489, 1948.
6. Lundgout, L.: Clin. Proc. Child. Hosp., 3: 269, 1947.

CRANIOFACIAL DYSOSTOSIS. (Revista Chilena de Pediatria, Santiago, 20: 337, Aug. 1949). Fuentes Acevedo and Valenzuela Lavin report the first case of craniofacial dysostosis in Chilean pediatric literature. A 13-year-old girl presented all the symptoms described by Crouzon as characteristic of the condition. There was no familial tendency. The authors studied the collaterals for three generations and did not find another instance of this syndrome. Early diagnosis is important in the surgical treatment of the condition. Operation in their case was not indicated because the patient was seen late, after the establishment of ocular lesions, and did not have intracranial hypertension. The cerebral and ocular lesions remained unchanged during the seven years the patient was observed. Neurosurgical intervention is indicated in cases with clinical symptoms, intracranial hypertension and roentgen signs of craniostosis.—*Journal A.M.A.*

THE MONGOLIAN SPOT*

DOUGLAS D. PERRY, M.D.
New York.

The Mongolian spot is still often regarded as characteristic of the Mongolian race. Of course, this concept is erroneous as it is not limited to any one racial group. It was in 1885 that Erwin Baelz, a German physician, discovered this spot to be present in most of the newborn Japanese infants whom he examined in a Tokyo clinic.

Although this spot is quite characteristic of the yellow race and exists in the pure white races and pure negro races as a rare anomaly, the name Mongolian spot is misleading. A more appropriate term is "congenital blue spot." It is rarest among the Ashkenasic Jews hailing from Poland, Russia and Hungary. It is also rare among the Spanish Jews with an increasing frequency in Jews from Asia and North Africa. The congenital blue spot is present in different racial groups and its color varies with the different races. It is said to be dark blue among the Japanese, slate grey in the Chinese, a blackish grey among the Polynesians and very black among the Negroes and Eskimos. It is greenish in the American Indians in general and pale blue in European children with brunet hair. In Turkey, out of 11,784 infants and children who were examined, the incidence was found to be 3.78 per cent.

Location. The congenital blue spot is most often found in the sacrolumbar region, but it may be present anywhere on the body. It has been observed on the legs, arm, shoulders and face. These spots may be round or oval in shape, single or multiple, isolated or joined. The size varies from a few millimeters to more than 5 centimeters in diameter. These blue spots are usually seen at birth, or shortly thereafter, and usually fade as the child grows older, being absent by the seventh or eighth year. Occasionally, these spots persist into adult life, raising a point of differential diagnosis between blue nevus, chloasma, lentigo spots and melanosarcoma. The blue nevus most resembles the congenital blue spot. They are identical histologically, but the dimensions of the blue nevus do not exceed the diameter of a bean and they are usually indurated and slightly elevated. Many parents take this spot for

*From the Department of Graduate Pediatrics, New York Medical College Flower and Fifth Avenue Hospitals.

a traumatic ecchymosis. However, the accentuation of the bluish tint by pressure of the finger prevents one from confusing the congenital blue spot with the trace of a blow received some days before.

Legends. Among the common people of Japan it has been considered a result of cotius during pregnancy (Adachi). Siguen Kagawa (1765) believed that the obi, or common belt, of the Japanese women, decomposed the blood of the mother, and this, stagnating, affected that part of the child lying closest to the abdominal wall, i.e., the sacral region. Shinsai (1846) thought it due to coitus during pregnancy, the heat of the semen decomposing the blood of adjacent portions of the child in such a way that contact with the air caused it to turn blue.

In parts of Argentina this spot is considered merely a sign of African blood. In Ecuador and Peru it is called "the family emerald." In these countries it constitutes, moreover, a sure sign of crossbreeding, and the expression "greenback" is such an insult that the possessor of a blue spot is very careful not to admit it.

In Turkey the women believe that if during pregnancy she gets hit on the abdomen, the baby will develop a blue spot on the corresponding part of the body. Most negro mothers look upon these spots as birth marks peculiar to their children and race.

Anthropology. According to Brennemann we should think of this pigmentation as a normal human characteristic, not a recurrence of a lost ancestral condition, i.e., atavism, as suggested by Bach, but the persistence in rudimentary form of what was once perhaps a more, widespread and functional layer of pigment such as exists in certain monkeys. Why this remnant should favor the sacral and adjoining regions, when there is no such tendency in monkeys, has never been explained. The frequent occurrence in other parts of the body would lead us to think of possible vestigial deep pigmentation in any location where epidermal pigment is normally most abundant, following a general law in both man and monkeys that epidermal and corium pigment is found more or less in the same proportion (Adachi).

We can no longer regard these blue spots as exclusive race characteristics. They should be given the same value as other racial trait—color, hair, features, etc. Their presence or absence in given cases leads to highly probable but not positive determina-

tion as to race or to degree of contamination. As regards medical legal aspects, these spots alone can not be considered to prove the presence of negro blood in any immediate sense.

Histology. In ordinary pigmentation the pigment granules are in the basal layer of the epidermis. In the colored races this pigmentation spreads throughout the epidermis with varying degrees of intensity. A second element is the pigment granules in the surface layer of the corium. In congenital blue spot there is a third element in the form of cutaneous melanoblasts arranged in bands parallel to the skin surface in the corium.

To see this spot in negroes and dark skinned people in general is often quite difficult, but the following suggestions may prove helpful: (1) Examine surface of skin obliquely. (2) Exert pressure on the skin where spot is likely to be found and then release the pressure quickly. The momentary blanching permits this pigmented spot to appear more clearly.

Etiology. Pregnant women, regardless of racial background, are subject to pigmentary changes such as darkening of the nipple areola, freckles, linea nigra in the midline of the abdomen and the external genetalia. The intensity of this pigment phenomenon varies directly with the degree of general pigmentation of the skin. Thus it is slight in blonds and marked in brunets. The blue spot appears about the fourth month of intrauterine life, a time when the pigment metabolism of the mother is undergoing marked activity. It is well-known that the adrenal glands undergo hypertrophy during pregnancy and the relationship of the adrenals to pigment metabolism is apparent in Addison's disease. The ovaries and the pituitary gland are also connected with metabolism during pregnancy but the exact relationship of these endrocine glands to the so-called Mongolian spot requires further investigation, but it is an interesting hypothesis.

CONCLUSION

This subject has received considerable attention in the foreign literature which is quite natural as the "congenital blue spot" occurs much more frequently in the South American countries, China and Japan, etc., but it is a subject which deserves more attention from the English speaking countries. There is considerable mate-

rial in this country due to the many racial mixtures present. After briefly reviewing some of the recent and past literature on this subject certain facts become apparent.

1. This condition must be fairly common among the dark skinned people of North America.

2. It is a benign condition present at birth located in the sacro-lumbar region usually, and disappears by the time the child is 7 or 8 years old.

3. When it persists to adult life it raises the question of differential diagnosis between blue nevus, chloasma, lentigo spots and melanosarcoma.

4. A more appropriate name is "congenital blue spot," as it is not characteristic only of the Mongolian race.

5. The role of the adrenals, ovaries and pituitary gland requires more extensive investigation.

6. It is a hereditary condition transmitted according to Mendel's law.

REFERENCES

1. Boisserie-Lacroix: The Mongolian Blue Spot in Bordeaux. J. de méd. de Bordeaux, 94: 355, June 10, 1933.
2. Ferreira, C.: The Mongolian Blue Spot at Sao Paulo, Brazil. Arch. de méd. d. enf., 25: 23, Jan. 1922.
3. Eyzaguirre, R.: The Mongolian Blue Spot in Peru. Arch. de méd. d. enf., 25: 19, Jan. 1922.
4. Apert, E.: Mongolian Sacral Blue Spot in a Bi-Half Breed. Arch. de méd. d. enf. 25: 295, May 1922.
5. Brennemann, J.: "Mongolian" Pigment Spots. Am. Anthrop., 1907, N.S. IX.
6. Ashmead, A.: Spots on Skin of Dark Races. J. Cutan. Dis., 1905.
7. Pariser, H. and Beerman, H.: Extensive Blue Patchlike Pigmentation. Arch. Dermat. & Syph., April 1949.
8. Matus, S.: The Mongol Spot in the Cape Colored. South African M. J., April 1941, p. 121.
9. Prien, E.: The Sacral Spot in Bengal. Science, 95: 431, April 1942.
10. Field, H.: The "Mongoloid Spot" in Turkey and Iraq. Am. J. Phys. Anthrop., June 1940, p. 119.
11. Chinese M. J. 44: 1050, 1930.
12. J.A.M.A., Dec. 21, 1940.

67-47 A 224th Street, Bayside.

CLINICAL-PATHOLOGICAL CONFERENCE

WILLARD PARKER HOSPITAL, NEW YORK

Meeting held February 21, 1950

DR. PHILIP MOEN STIMSON, presiding.

WATERHOUSE-FRIDERICHSEN SYNDROME

DR. GLENN presented Case No. 172, a 3-year-old white female, admitted in extremis about 9:30 P. M. on January 14, 1950 and died 45 minutes later.

This patient was apparently well until about 10:30 A. M. on the morning of admission when she vomited twice and the mother noticed that she was a little irritable (temperature 99.6° F. at that time). The child, nevertheless, continued to play until about 4:00 P. M. when she asked to be put to bed. The doctor came shortly thereafter, advised the family to call a pediatrician. He gave no medication.

At 6:30 P. M. the mother noticed that a few bluish spots were appearing over the body and temperature was 104° F. By the time the pediatrician arrived, there were more blue spots, and pediatrician brought the child directly to this hospital.

Past History. No previous upper respiratory infection.

Physical Examination. Child was moribund, thrashing about on the stretcher, screaming, at first coherent phrases and in a few minutes quite incoherent. Her entire body was heavily scattered with ecchymosis ranging from the size of a quarter to pinpoint petechiae.

The right ear drum was red; the throat moderately red. A few râles were heard in the chest anteriorly.

Heart rate was 118, sounds of fair quality.

The neck was quite stiff. Brudzinski and Kernig positive. Babinski negative.

The following therapy and procedures were done in order mentioned (Protein 32 mg.; sugar 98, Neisseria meningitidis): (1) Lung puncture: Fluid clear, Pandy negative. No cells. (2) Sodium sulfadiazine, 1.0 gram. (5 per cent solution) intravenously. (3) Aqueous adrenocortical extract, 20 cc. intravenously. (4) Placed in oxygen tent. 10cc. subcutaneously.

The patient went rapidly downhill, more ecchymosis and petechiae appearing during the examination and treatment. She became more disoriented, then spoke no words, responded to no stimulation. Extremities became cold and cyanotic from nails upward, and just after being placed in oxygen tent, the patient expired.

Discussion. DR. STIMSON. The onset was at 3:00 P. M. Death occurred 6¼ hours after onset.

WATERHOUSE-FRIDERICHSEN SYNDROME

DR. FURMAN presented Case No. 58, a 16-month-old colored boy, who was transferred to Willard Parker Hospital on January 5, 1950 from Harlem Hospital with a diagnosis of "possible poliomyelitis." He had a history of an upper respiratory infection three weeks before the present illness. He had developed a high fever on the morning of January 5, 1950, was irritable and vomited once. On the afternoon he appeared to be unable to stand and a questionable, faint rash was noted on the face.

On admission at about 12 midnight, he had a temperature of 100.2° F., appeared drowsy, but in no acute distress. There was questionable neck stiffness, absent knee jerks and ankle jerks and questionable weakness of both lower extremities. A spinal tap revealed clear, colorless fluid with no cells.

The child was put to bed. His respirations were rapid, and he passed several loose stools containing undigested matter. At 4:45 A. M. his breathing was a bit slower. At 5:00 A. M. he was found dead.

Necropsy. DR. DOLGOPOL presented the gross specimens.

The first child (girl, 3 years old) showed a number of petechiae over the trunk and in the conjunctivae of both eyes. The heart was rather large (110 grams), but normal in shape. The lungs were congested. The liver was pale and somewhat enlarged. The spleen was congested and enlarged, with prominent follicles. The pulp scrapes moderately. Each adrenal weighed 10 grams. Both were dark red in color, with blood oozing from the cut surface. The kidneys were slightly congested. A few petechiae were present on the serosa of the small intestine. The brain weighed 1,270 grams (heavier than normal), was edematous and soft. The meninges also were edematous, but there was no subarachnoid exudate.

Meningococcus has been isolated from the spinal fluid antemortem and from the postmortem heart's blood culture.

Microscopic slides were projected.

Heart: The heart showed a minimal focal myocarditis with polymorphonuclears in some foci. The valves were intact. In the adrenals the hemorrhages involved the entire cortex and in places spilled into the medulla. There was no meningitis.

The second patient had been in the hospital less than 24 hours, and the case became one for the medical examiner. The autopsy was performed 36 hours after death. The spinal fluid was sterile. No blood culture has been taken. No report on postmortem material was received by the medical examiner at the time of this conference. The body showed some hemorrhagic rash on the face, but not on the body.

Brain was congested, but there was no meningitis. The heart appeared normal. Mucus was present in both main bronchi. The lungs were congested. The liver, spleen and kidneys showed nothing remarkable. Both adrenals were hemorrhagic. A few petechiae were present on the intestinal mucosa.

Microscopic Findings. Cortical hemorrhages were present in both adrenals. Minimal focal myocarditis was present. No meningitis was found.

The pathology of both cases was discussed after the presentation of histories.

The second patient was a medical examiner's case because the patient had been in the hospital less than 24 hours, and the autopsy was performed in Bellevue Hospital about 36 hours after death.

There were several small hemorrhagic papules on the face but not on the body. No sample of the rash could be taken from the facial lesions for microscopic study. The brain was very much congested but there was no meningitis. Most important findings were hemorrhages into the cortex of both adrenals. There was a marked congestion and edema of both lungs. The main bronchi contained much mucus, but their lumen was not entirely obstructed. All organs were congested. Microscopically, a minimal focal myocarditis was found. All organs were markedly congested.

Discussion. I do not have very much to show. Autopsy was started 36 hours after death. The tissues were not very satisfactory. Both adrenals presented the same case. Not either brain showed any meningitis.

Microscopic Findings were projected by DR. DOLGOPOL. In this case there were scattered red cells. The only reason for a lumbar puncture is if one suspects a meningitis. The presence of one infection does not rule out the presence of another. Those are the cases that so frequently have both manifestations.

TUBERCULOUS MENINGITIS

DR. JUROW presented Case No. 4784, an 8-year-old colored boy, who was admitted to Willard Parker Hospital for the first time on April 8, 1948 at the age of 6 years. He had been hospitalized at Greenpoint Hospital since March 29, 1948 for tuberculous meningitis, miliary tuberculosis and tuberculous peritonitis and had been treated with intramuscular and intrathecal streptomycin.

On Admission he showed slight meningeal signs, a distended abdomen and signs of pulmonary involvement. His chest x-ray showed infiltration of the right upper lobe and mediastinal adenopathy. Spinal fluids showed increased cells and protein and low sugar. Streptomycin, 2 grams intramuscularly, and 100 mg. intrathecally, were begun on admission.

The patient showed gradual, clinical improvement. On June 10, 1948 the intramuscular streptomycin was cut to 0.5 grams twice daily. His spinal fluid sugar became normal on about July 1, 1948, and on July 7, 1948 intrathecal streptomycin was stopped. He continued to run a low grade fever till the end of August 1948. Intramuscular streptomycin was stopped on September 15, 1948 and patient was discharged free of symptoms and with signs of slight central damage (positive Romberg sign, ptosis of the right eyelid, dilation of the right pupil and right external strabismus).

The boy was well, except for occasional upper respiratory infections and did not return to Willard Parker Hospital until March 25, 1949 when he was admitted with a history of headache and listlessness for about one week. He showed no meningeal signs and his spinal fluid was normal. He was discharged on March 29, 1949.

He was re-admitted (third admission) on May 18, 1949 with a history of headache and vomiting during the previous week. There were no meningeal signs and several spinal taps produced normal fluid. The child was febrile, became increasingly lethargic and developed meningeal signs, about a week later. On June 4, 1949 his spinal fluid showed a low sugar, and streptomycin, 1 gram intra-

muscularly, daily, was begun. He showed little improvement and on June 17, 1949, intrathecal streptomycin, 50 mg. daily, was resumed. In the following weeks, the patient developed lateral nystagmus. He showed gradual improvement and clearing of his meningeal signs. On August 1, 1949 intrathecal therapy was stopped and on September 6, 1949 intramuscular streptomycin was also stopped. He was discharged on September 15, 1949, free of signs of active disease.

4th Admission. The child was re-admitted on November 21, 1949 with a history of fever and cough for one week. On admission he showed impaired hearing, strabismus and positive meningeal signs. Streptomycin, 1 gram intramuscularly, daily, and 50 mg. intrathecally, every other day, and promizole, 1.5 grams daily were begun. His spinal fluid showed increased cells and protein and low sugar. Acid-fast bacilli (sensitive to streptomycin) were cultured from the fluid. X-ray of his chest showed no changes from the pictures seen on the first admission. He showed some clinical improvement, but his spinal fluid sugars remained low and he continued to run a low grade fever. Intrathecal streptomycin was increased to 50 mg. daily on December 12, 1949 and on January 13, 1950 to 100 mg. daily. The child continued to go downhill and did not take his feedings well.

On January 17, 1950 the patient's temperature spiked and on the next day a morbilliform eruption and Koplik's spots were noted. He was unresponsive and was maintained on parenteral fluids. On January 18, 1950 he had a generalized convulsion and after this lapsed into coma. His pulse became feeble, his respirations shallow. No reflexes were obtainable. He was treated with oxygen, pencillin, continuous suction and fluids intravenously via a cut-down. In the next few days, he improved somewhat and could be fed by gavage. His temperature rose again on January 26, 1950, his respirations became weaker and his pulse faint. Intramuscular streptomycin was maintained throughout but the patient was given no intrathecal therapy during his last week of life. He died on January 29, 1950.

X-rays were presented by Dr. Schwartz. There were about 14 different examinations.

Necropsy. Dr. Dolgopol presented the gross specimens. No active tuberculosis was found in the lungs, but two tiny encapsu-

lated foci, about 2 mm. in diameter were found, one on the anterior aspect of the right upper, the other on the lateral aspect of the left upper lobe. A few small caseous, partly calcified lymph nodes, were found on the left side of the trachea. The heart was large and weighed 150 grams. Normal for his age would be 100 grams. His kidneys also were large, weighing 120 and 190 grams instead of weighing about 70 grams each. They were pale-pinkish in color and appeared to be degenerated. A few hemorrhages were present in the pelves. The ureters were normal. The urinary bladder contained a bloody urine. The liver also was somewhat enlarged.

Brain: The surface was rather pale. No trace of meningitis could be seen on the convexity of the brain. A cloudy gelatinous exudate was present at the base of the brain, mainly around the chiasm. The roof of the fourth ventricle was not adherent to the cerebellum. All ventricles, however, were somewhat distended and the aqueduct was narrow. The meninges about the cervical and thoracic cord were thickened.

Microscopic Slides. The meninges in the region of the chiasm were edematous, contained a few fibrotic nodules, probably healed tubercles and showed a lymphocytic infiltration. The anterior cerebral arteries were thick-walled, with a very narrow lumen. As a result of poor circulation there was a subependymal softening of brain tissue along the floor of the anterior horn. Marked proliferation of subependymal glia was present not only in the lateral ventricles, but also in the aqueduct. A good deal of edema and many lymphocytes were present in the meninges of the medulla and the cord. One healed tubercle was found in the meninges of the medulla, another in the meninges of the cervical cord and a small epithelioid tubercle was found in the thoracic meninges.

The focus of the left lung was completely arrested. In the right, numerous bronchi showed bronchiectasis and there was some interstitial fibrosis. Otherwise, lungs were markedly edematous.

Heart: There were several foci of plasma cells indicating the presence of focal myocarditis. Patient recently had measles and although myocarditis is not frequent in that disease, measles could be the cause of myocarditis in this case rather than tuberculosis.

The liver shows evidence of passive congestion.

The kidneys here and there showed some evidence of old glomerular damage. In addition, widespread hyaline-droplet de-

generation could be seen in the tubules. Casts were present but no blood was seen in the collecting tubules. There was no evidence that tuberculosis played any part in this renal disease. There were no healed tubercles anywhere. The kidneys were not normal for some time. Urine contained some blood, but it was apparently due to hemorrhages in the pelves.

Tuberculosis was to a great extent cured. This is the best healed tuberculous process in the meninges that has come to autopsy in our material.

Death was probably due to the renal disease which became cardiorenal as a result of focal myocarditis. It is possible that the measles not only caused the myocarditis, but that it also increased the tubular degeneration.

Acute Painful Torticollis and Cervical Subluxation. (Acta Chirurgica Scandinavica, Stockholm, 98: 212, July 4, 1949). Sulamaa reports 2 girls with painful torticollis. The first child, aged 6, had a painful torticollis when she awoke one morning. When she was brought for treatment two months later, roentgenograms indicated subluxation between the second and third cervical vertebrae. The pain ceased immediately and the neck could be freely moved when the head was hung in hyperextension. The plaster collar which had been applied was removed after one month, and the girl appeared to be cured. Three days later, however, the condition suddenly recurred and disappeared again when the head was placed in a position of hyperextension. Recovery was final after treatment with a plaster collar for three months. In the second case painful torticollis occurred after otitis of long duration. Roentgen examination performed with anesthesia showed subluxation between the second and third cervical vertebrae when the head was flexed. Dislocation could not be established in hyperextension. As in the first case, the pain ceased and the contraction loosened when the head was hung in a position of hyperextension. Recovery followed treatment with a plaster collar for two and a half months. The author assumes that in the first patient an unnoticed trauma was probably the cause of the acute torticollis. In the second case there was probably an infectious cause.—*Journal A.M.A.*

DEPARTMENT OF ABSTRACTS

KENDIG, E. L. AND GUERRY, D.: THE INCIDENCE OF CONGENITAL IMPATENCY OF THE NASOLACRIMAL DUCT. (Journal of Pediatrics, 36: 212, Feb. 1950).

In a study of 1,000 consecutive, unselected, full-term newborn infants, 57 (5.7 per cent) cases of congenital impatency of the nasolacrimal duct were found. These were evidenced by the presence of epiphora and the appearance of mucopus after pressure over the affected lacrimal sac. The majority of the cases clear up after conservative treatment which consists of local application of a penicillin ointment containing 1,000 O.U. per gram of ointment base three times daily, and massage by the mother over the region of the sac. If the condition does not clear up in six months then probing of the nasolacrimal duct is advised. In those cases in which probing must be carried out, it should be done with the patient under general anesthesia and not as an office procedure. The procedure which was found to be most satisfactory was the introduction of the No. 1 Bowman probe through the superior punctum and canaliculus after dilatation of the punctum with the dilator, with the patient under ether anesthesia. The inferior meatus is explored with a submucous elevator and the probe located and rubbed until bare metal is felt.

MICHAEL A. BRESCIA, M.D.

HUTCHISON, J. H.: CONGENITAL TOXOPLASMOSIS. REPORT OF TWO CASES. (Archives of Disease in Childhood, 24:303, Dec. 1949).

Human toxoplasmosis may take five forms: 1. A congenital infection, possibly transmitted via the placenta less probably reaching the amniotic fluid from the vagina. 2. An acquired acute encephalitis in older children. 3. An acute toxoplasmosis in adults with fever, pulmonary signs, and sometimes with a diffuse maculopapular rash indistinguishable clinically from tick typhus. 4. A chronic encephalitis in adults and 5. A symptomless infection in adults out of which presumably the first type arises during intrauterine life. The most common clinical features of the congenital type are hydrocephalus, intracranial calcification, bilateral macular

choroido-retinitis and disturbances of nervous function. The only certain proof of toxoplasmosis is the recovery of the toxoplasma from the body fluids, especially from the cerebrospinal fluid. This, however, is usually unsuccessful. Hence, tests for determining toxoplasma-neutralizing antibodies in serum have been devised and used to establish infection with the toxoplasma.

MICHAEL A. BRESCIA, M.D.

SCHNITKER, M. T.: A SYNDROME OF CEREBRAL CONCUSSION IN CHILDREN. (Journal of Pediatrics, 35:557, Nov. 1949).

Minor head injuries are of common occurrence in children during active play in which the child falls to the ground or against some object. Occasionally such a minor injury is followed by a delayed period of unconsciousness so as to cause anxiety that actual intracranial damage may be present. The syndrome as described by the author is as follows: A child at play falls to the ground or against an object and bumps his forehead region. This is followed by a momentary dazed state and usually crying as a natural reaction. Then follows a lucid interval, lasting up to two hours, during which the child shows mild shock with pallor, slight slowing of the pulse, irritability and vomiting. The child is made to lie down and then becomes increasingly drowsy and falls alseep. During this period the child is more difficult to arouse. When seen by a physician, the sequence of events—head injury, lucid interval, stupor—readily suggests the possibility of extradural hematoma but the neurological examination is otherwise normal. It should be noted that extradural hematoma in children is unusual (Beekman) and when present is usually accompanied by severe shock and anemia (Campbell). The delayed stupor or semicoma in concussion continues from three to six hours and usually carries the child into and through the night. He arises the next morning fully recovered without any sequelae.

MICHAEL A. BRESCIA, M.D.

EARLE, A. M.: METHYL TESTOSTERONE AND PLASMA FOR PREMATURE INFANTS. (Journal of Pediatrics, 36:87, Jan. 1950).

Thirty-six prematures were divided into two groups, the first containing 24 cases and the second 12 cases. In the later group

one premature died on the seventh hospital day with aspiration pneumonia. Both groups were given vitamin K daily for three days, a hypodermoclysis of 50 to 100 cc. of 2½ per cent glucose once or twice daily for one week depending on skin turgor and weight. After the first week, vitamins C and D were added and during the third week ferrous sulphate was added. After the first 48 hours the infants were placed on a stock evaporated milk formula of 1 to 2 ounces every three hours. Those in group I were given 0.5 cc. of crude liver extract every other day. Those in group II were given 5 mg. of methyl testosterone in the 10:00 A.M. feeding daily. In addition, those in group II received 100 to 150 cc. of plasma orally daily in divided amounts until they attained the weight of 2,722 Gm. In group I the average daily weight-gain was 9 Gm. and the average hospital stay was 58 days and the number of days necessary to regain the birth weight varied from five to 54 days, averaging 14 days. In group II (those receiving testosterone and plasma) the average daily weight gain was 22.5 Gm. and the average hospital stay was 25 days. The number of days to regain birth weight varied from zero to five days, averaging two days. No untoward symptoms developed as a result of the use of the testosterone.

MICHAEL A. BRESCIA, M.D.

BAKWIN, H.: PSYCHIC TRAUMA OF OPERATIONS. (Journal of Pediatrics, 36:262, Feb. 1950).

Tonsillectomy and adenoidectomy still remain the commonest operative procedures in children. This is usually the child's first admission to a hospital and is often the first painful separation of the child from the home. It is no wonder then that these children suffer a certain amount of psychic trauma and a number of cases of fears, anxieties and other symptoms have followed an operative procedure. To ease the plight of the child who is to be operated on, it is suggested to use the following measures: 1. Give the child a forthright and honest explanation of the reasons for the operation. 2. Try to avoid the element of surprise and confusion at the hospital. A picture of the hospital, the operating room, white gowned nurses and doctors should be described. 3. Explain that the purpose of the anesthesia is to spare the child pain during the operation. Assurance must be given that this forced sleep is

temporary and will be followed by complete awakening and survival. 4. Emphasize to the parents the need for constant reassurance and affection in the postoperative period. 5. Whenever possible, the doctor personally should lead the child from the mother to the operating room. 6. Permit overt manifestation of postoperative hostility, rather than suppress it.

MICHAEL A. BRESCIA, M.D.

GAIRDNER, D.: THE FATE OF THE FORESKIN. A STUDY OF CIRCUMCISION. (British Medical Journal, 4642:1433, Dec. 24, 1949).

Circumcision is one of the oldest operations, one of the most common procedures and yet has been accorded the least critical consideration. It is well to remember that the prepuce is still in the course of developing at the time of birth, and the fact that its separation is usually still incomplete renders the normal prepuce of the newborn non-retractable. At birth about 4 per cent have a fully retractable prepuce, at 6 months about 20 per cent are retractable, at one year about 50 per cent, at two years about 80 per cent and by three years 90 per cent have a completely retractable prepuce. The prepuce helps to protect the glans of the incontinent infant. Although circumcision is considered a simple and innocuous procedure, nine deaths were reported in England and Wales for 1947 as a result of circumcision in infants under a year of age. At various times circumcision has been advised for the treatment or prevention of various diseases, viz, phimosis, balanitis, enuresis, paraphimosis, venereal diseases, penile cancer and cervical cancer in women. However, the author questions the validity of the value of circumcision in the above named conditions. In the light of these statements a conservative attitude towards the prepuce is proposed and a routine for its hygiene is suggested. If adopted this would eliminate the vast majority of the circumcision operations performed annually along with their yearly toll.

MICHAEL A. BRESCIA, M.D.

HEALTH AND LONGEVITY AT THE MID-CENTURY. (Statistical Bulletin, Metropolitan Life Insurance Company, 31:1, Jan. 1950).

The drop in mortality from the principal communicable diseases of childhood has been nothing short of spectacular. For the four

diseases combined—measles, scarlet fever, whooping cough and diphtheria—the death rate at ages 1 to 14 years declined from 171.8 per 100,000 policyholders in 1911 to 2.6 in 1949. Forty years ago, the mortality from diphtheria alone was more than 30 times the present rate for all four diseases combined. One of the unfavorable features of the year (1949) was the high incidence of acute poliomyelitis. More than 42,000 cases were reported in the general population, as compared with less than 28,000 in 1948. Substantial progress has been made against rheumatic fever, which is the leading cause of heart disease from early childhood up to about 45 years of age. Among industrial policyholders from ages 5 to 24 years, the death rate from rheumatic fever and from organic heart disease, which is usually of rheumatic origin at these ages, dropped more than three-quarters between 1911-1915 and 1944-1948. The downward trend in mortality appears to be due to a lessening in severity of the disease and possibly to a reduction in the occurrence of new cases. The decline in the incidence of rheumatic fever is believed to reflect better child care, including the use of antibiotics in the treatment of streptococcal infections.

MICHAEL A. BRESCIA, M.D.

NEGATIVELY REACTING CHILD CONTACTS AND BCG VACCINE. (Tubercle, London, 30: 218, Oct. 1949.) Shaw and Wynn-Williams carried out a survey of cases of tuberculosis registered in the County of Bedfordshire, England, from Jan. 1, 1949, to June 30, 1949. One hundred and fifty-nine patients with pulmonary tuberculosis had contacts in the age group up to 14 years. Two hundred and fifty-three of the total of 266 contacts (95.6 per cent) were tuberculin tested. The conclusions from these tests were grouped according to the sputum status of the respective source cases of the contacts and tabulated with particular reference to the number of negative reactors. Ten new contacts of fresh sputum-positive source cases will be found nonreactors every three months. Fifty-two negative reacting contacts of 100 sputum-positive cases now require BCG vaccination. It is contended that all contacts of pulmonary tuberculosis in the age group up to 14 years who are negative reactors should be given BCG vacination.—*Journal A.M.A.*

Do you know...

a bride...

or a graduate...

or a new baby...

or a birthday girl?

Chances are you're trying to dream up the perfect gift for one of these lucky people!

And what gift could be more wonderful for any or all of them—than a crisp U.S. Savings Bond!

Remember U.S. Savings Bonds pay $4 for every $3 at the end of 10 years.

U.S. Savings Bonds do not lose their value if they're lost, stolen or destroyed.

They can be turned into cash in case of emergency.

Settle your gift problems at your bank or post office—with U.S. Savings Bonds! While you're at it—how about some for yourself!

Automatic saving is sure saving—U.S. Savings Bonds

Contributed by this magazine in co-operation with the Magazine Publishers of America as a public service.

He's heard the call for

VI-DAYLIN

TRADE MARK

(Homogenized mixture of Vitamins A, D, B_1, B_2, C and Nicotinamide, Abbott)

And that's one call he'll answer—come hill or high fences. For VI-DAYLIN's pleasing, citrus-like flavor and odor make a hit with children every time. A glance at the formula reveals the sound vitamin therapy in this sparkling yellow liquid. One 5-cc. teaspoonful is the average daily dose for children up to the age of 12. They like it direct from the spoon. For infants, VI-DAYLIN mixes readily with milk, fruit juice or cereal. For grown-ups who dislike tablets, capsules or unpleasant-tasting preparations, VI-DAYLIN is a refreshing alternate. It is stable at room temperature, has no fishy odor. Available at all prescription pharmacies in 90-cc., 8-fluidounce and 1-pint bottles. Abbott

Each 5-cc. teaspoonful of VI-Daylin contains:	
Vitamin A	3000 U.S.P. units
Vitamin D	800 U.S.P. units
Thiamine Hydrochloride	1.5 mg.
Riboflavin	1.7 mg.
Ascorbic Acid	40 mg.
Nicotinamide	10 mg.